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ALVEOLAR CELL CARCINOMA*

CHARLES R. KESSLER, M.D.

Birmingham, Alabama

Alveolar cell carcinoma is a primary malignant tumor of the lung which has been diagnosed with increasing frequency each year for the past ten years. In spite of the fact that the first record of such a tumor dates back to 1876 there remain many points which need clarifying. There is no consensus as to its proper name, cause, origin or treatment. The tumor has been variously called papillary gelatinous adenocarcinoma, primary alveolar cell tumor, pulmonary adenomatosis, mucocell papillary adenocarcinoma, diffuse epithelial hyperplasia, diffuse primary alveolar epithelial carcinoma, malignant adenoma, cystic papillary lung tumor, muco-epithelial hyperplasia, *etc.*

It is commonly thought to be of multicentric origin but some recent reports have cast doubt as to this point. It is suggested that perhaps the apparent multicentricity of the tumor is caused by air borne metastases rather than multiple points of origin. The similarity between this tumor in man and an epidemic disease in sheep called jaagsiekte has stimulated thought that perhaps this (and maybe even all neoplastic disease) is caused by an ultra microscopic agent such as a virus. It is certainly true that the epidemic disease in sheep produces in the lungs of these animals pathologic changes which are essentially indistinguishable from what we call alveolar cell carcinoma in humans. Though the disease in sheep does occur in epidemic proportions it has never been experimentally transmitted from one animal to another.

Many agents have been known to stimulate hyperplasia and metaplasia in the pulmonary alveoli in humans but none has been definitely incriminated as a cause of alveolar cell carcinoma. Attempts have been made by such people as Dr. Jonas Salk to transmit the tumor found in humans to experimental animals—but without success. Drs. H. G. Grady and H. L. Steward produced malignant tumors of alveolar origin by injecting dibenzanthracene in experimental mice. All of these various attempts at production of the tumor or transmission of the tumor have given us no concrete conclusions, however.

The pathology of alveolar cell carcinoma is not like that of any other pulmonary neoplasm. The tumor is present in three forms grossly: (1) as a multiple nodular growth distributed throughout one or both lungs; (2) as a diffuse form involving uniformly one lobe or more; (3) as a focal disease in a limited area. The diffuse form shows evidence grossly and microscopically, suggesting that it is the result of coalescence of a multiple nodular form. The nodules vary in size from miliary to grape size and usually are grayish white or pinkish in color. The more diffuse form resembles a pneumonia or atelectasis and the focal form most nearly resembles a tuberculoma. It is reported that pleuritis with fibrinous or serous exudate and adhesions usually are present but in at least eight or nine cases in this report no appreciable evidence of pleuritis was found. It is likewise reported that cavities and bronchiectasis are rare but again two of the nine cases presented showed definite cavity lesions both grossly and by x-ray and one of the nine showed bronchiectasis demonstrable on bronchography. Metastases of the tumor are usually late in the disease and may not be found at all if the case is one of extensive diffuse involvement. In such cases the patient usually dies of asphyxia before

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metastases have occurred. In the more localized disease to the brain, regional lymph nodes, liver, adrenals, bone, etc. have been recorded. Microscopically the prominent feature is that the intra-alveolar septa are lined with—but not invaded by—cells which vary from high cuboidal to columnar in type with all degrees of proliferation and with some papillary projection into the alveolar lumen. The cytoplasm is clear and may be pink stained because of a large amount of mucous. There is marked pleomorphism in the size of the cells and the nuclei.

As stated earlier there is still much difference of opinion as to the origin of these cells as to whether they come from a true alveolar lining epithelium, from the basal cells of the terminal bronchioles or from the so-called mesenchymal septal cells. As we all know, for many years there was an argument as to whether any alveolar epithelium persists beyond prenatal life; there is considerable evidence recently that either there is a true epithelial lining of the alveoli or at least there are isolated cells left after this disintegration of part of the membrane by rapid ingrowth of blood vessels between the alveoli.

Clinically there is a slight predominance of males over females though this predominance is not nearly as marked as in the bronchiogenic carcinomas. In the small series of nine cases reported here there were five females and four males. There is no consistent relationship to smoking. In many patients no symptoms whatever are present and the lesion is found simply on routine chest survey. Typically, patients do have cough productive of clear mucoid sputum which will vary from a few cubic centimeters to over a liter in 24 hours. Dyspnea becomes a feature in many of the patients as more lung is involved. Weight loss and fatigue are not prominent early symptoms; nor is cyanosis, hemoptysis and chest pain. Once symptoms such as dyspnea and the productive cough develop they are usually progressive to a fatal end with the rapidity varying from case to case. The physical signs are dependent on underlying involvement as to whether it is a diffuse process or is limited to a single lobe. The blood picture has not been remarkable in these cases. Sputum examinations for tumor cells will report positive findings in a high percentage. X-ray appearance is seldom pathognomonic of alveolar cell carcinoma and in most instances the lesion cannot be differentiated from ordinary bronchiogenic carcinoma, metastatic carcinoma, tuberculosis or sarcoidosis. Bronchoscopy and bronchography are usually of little value except that in some cases positive bronchial washings can be obtained. Direct evidence of alveolar cell carcinoma is not seen in the major bronchi. The diagnosis of alveolar cell carcinoma is very difficult to make during life and can be established with certainty only by tissue diagnosis from resected portions of the lung. Occasionally a late case can be diagnosed by lymph node biopsy. It is probably as true in alveolar cell carcinoma as in other carcinomas that the time to make the diagnosis is when the disease is so early that an exact diagnosis can be made only by study of resected tissue.

The treatment of alveolar cell carcinoma has not been satisfactory by any means. The tumors are notoriously resistant to x-ray. Nitrogen mustard and T.E.M. have not appeared to be of any benefit. The only cure, as in other lung cancers, has been following resection. In the advanced cases in which there are copious amounts of sputum nothing has been of avail in reducing the terrific bronchorrhea. The treatment of alveolar cell carcinoma is surgical with the amount of lung tissue to be removed depending on the findings. If the lesion appears to be limited to a lobe, probably lobectomy is sufficient though there are advocates of total pneumonectomy for all cases. A review of the literature has not shown that more extensive resections have been productive of a greater number of cures. Segmental resection is probably never advisable. One thing has been striking both in cases presented here and in reviewing the literature on the surgical treatment of alveolar cell carcinoma: Patients who are resected either have recurrences in the same or contralateral lung in a matter of a few months or they apparently go for many years without further trouble.

CASE REPORTS

Case 1. The first case operated on was a 62-year-old white female who reported a history of attacks of "virus pneumonia" in March 1945, February 1947 and June 1947. Between the attacks of pneumonia the patient had a cough productive of 4 or 5 drams of white foamy sputum in 24 hours. The cough and expectoration were worse on going to bed at night and on arising in the morning. There was no chest pain, wheezing or hemoptyses. Dyspnea on exertion was mild. X-rays in March

1945 through June 1947 showed "pneumonitis, left lower lobe." Bronchoscopy done in June 1947 showed mucoid secretions of the left lower lobe which were negative for tumor cells. Bronchograms made in 1948 showed some mild cylindrical bronchiectasis in the left lower lobe and lingula of the left upper lobe. At this time sputum was somewhat purulent and washings showed fat laden macrophages but no tumor cells. Surgery was recommended because of the recurring pneumonitis and the diagnosis of bronchiectasis. For reasons of her own the patient deferred surgery until November 1948 at which time thoracotomy was done. The lower lobe and lingula were rather small and appeared atelectatic. There were no adhesions. The lower lobe and lingula were removed by individual ligation technique and the postoperative course at first was uneventful. The pathologic report was typical pulmonary alveolar cell carcinoma called adenomatosis at that time. Six weeks after surgery the patient began to raise large amounts of foamy sputum and x-ray a week later showed nodular infiltration in the lower part of the left upper lobe and in the right base. The amount of sputum increased and by February 22nd the patient raised 6 to 8 ounces of thin watery sputum in 24 hours. Dyspnea began to be a problem at that time. Patient's symptoms of cough, sputum and dyspnea gradually increased and there was further progression of x-ray evidence of lesions in both lungs. X-ray therapy was attempted but no improvement ensued. Patient expired in June 1949 of respiratory failure. Autopsy was not permitted.

Case 2. This 63-year-old white female complained of a chronic cough and chronic sinusitis for about 15 to 18 years. She was first seen March 16, 1951 and stated that beginning in October 1950 her symptoms had become much worse. The cough previously had been largely non-productive but at the time she was seen she was raising 2 to 3 ounces of white mucoid sputum during the day. There was no chest pain and no hemoptysis; there was about a 15-lb. weight loss and no fever. The patient was rather poorly nourished and had a constant dry non-productive cough. There was no clubbing or cyanosis of the fingers. X-ray showed generalized mottled infiltrative lesions throughout both lung fields and the lesion did not appear to be confluent in any area. The patient had previously been studied with sputum examinations for acid fast bacilli and fungi which were negative. Tuberculin and histoplasmin were negative. Bronchoscopy was done March 17th at which time only some generalized redness and a small amount of mucoid sputum was seen. Bronchial washings were reported as showing definite tumor cells probably from an alveolar cell carcinoma. The patient had a progressive downhill course in spite of nitrogen mustard therapy and died approximately 2 months after first being seen. A definite tissue diagnosis was not made but this case seems to fit the typical multiple nodular type.

Case 3. This was a 56-year-old white male who had been employed all of his life as a coal miner. He had been admitted to the hospital in July 1952 because of weakness, weight loss and cough. Physical examination was not remarkable but x-ray of the chest showed an annular lesion in the lower portion of the left lung field. The initial impression was that this was a bronchiogenic carcinoma with necrosis in the center. Bronchoscopy and study of bronchial washings were not remarkable. On August 4, 1952 exploratory thoracotomy was done and a left lower lobe lobectomy was performed. Postoperatively the patient had no difficulty and the upper lobe expanded normally. There was no lymph node involvement. Pathologic report on this specimen was first that it was a metastatic carcinoma from the colon. Complete body survey showed no primary elsewhere and the pathologic diagnosis was later changed to alveolar cell carcinoma with a large amount of mucous production. This patient has remained completely well and his last x-ray in July 1956 showed no sign of tumor.

Case 4. This was a 44-year-old white male who was first seen in April 1953. This patient was asymptomatic and on a routine survey was found to have a mass in his right lung. Physical examination was negative except for some palpable nodes in the supraclavicular area. Bronchoscopy was done and was negative. Biopsy of the supraclavicular nodes was negative except for reticuloendothelial hyperplasia. On April 30, 1953, exploratory thoracotomy was done at which time the mass was found to involve the upper lobe and the middle lobe. An upper and middle lobe lobectomy was performed. The patient had no difficulty in the immediate postoperative period. Permanent sections were reported as alveolar cell carcinoma, grade 3, with no involvement of the lymph nodes. The patient remained well until October 1954 at which time he began to notice a cough which on occasion would be productive of a large amount of phlegm. He had some wheezing and fatigability and some weight loss. Examination at that time showed a 20-pound weight loss with lymphadenopathy in the right supraclavicular area and in the right axilla. X-ray of the chest showed bilateral nodular disease suggesting a recurrence or metastatic carcinoma. A biopsy of one of the lymph nodes showed a rapidly growing malignancy. The patient was given nitrogen mustard with no benefit and died in December

1954. Autopsy showed extensive carcinomatosis of the lungs, mediastinal lymph nodes, superior mediastinal nodes, pericardial cavity, heart, liver, stomach, peritoneal cavity and adrenals.

Case 5. This was a 65-year-old white female who had been a diabetic for many years. She was first seen in May 1953 because of a circumscribed lesion in the lower portion of the right lung field. This lesion was first seen by x-ray in 1949 and annual x-rays thereafter showed no change until four years later. At this time the mass increased in size. The patient denied any symptoms referable to her chest whatever. Physical examination was not remarkable and except for the diabetes the patient appeared to be well. Patient did not react to histoplasmin or to tuberculin and thoracotomy was recommended. The lesion appeared to be limited to the right middle lobe and a right middle lobe lobectomy only was done. The upper and lower lobes appeared to be grossly normal and there was no lymphadenopathy. The report on the resected specimen was alveolar cell carcinoma, grade 1. This patient had considerable difficulty in the early postoperative period with chest wall pain which she tolerated very poorly. She eventually cleared and was discharge from our care. She did not develop any respiratory symptoms. However, in the spring of 1955, some 2 years after her original surgery, she had a pathologic fracture of the hip and x-rays at that time showed evidence of recurrent tumor in the lungs. The patient died July 10, 1955 presumably of recurrent and metastatic disease. An autopsy was not granted.

Case 6. This was a 50-year-old white male seen August 10, 1954. His illness began in January 1954 with what was called "flu." X-ray of the chest a short time after this episode demonstrated some infiltrate in the left upper zone. The lesion persisted to the time we first saw him with perhaps some remissions and progressions. It was known that a chest x-ray one year earlier was normal. The patient had no symptoms except a "cigarette cough" in which he denied any change. There was some aching pain in the left shoulder and left arm. He had had some recurring bouts of low grade fever but no weight loss, no hemoptysis and no wheezing. Physical examination showed clubbing of the fingers which had been noted by the patient's wife about 6 months earlier. There was a palpable node in the left supraclavicular area but otherwise physical examination was normal. X-ray showed a soft infiltrate measuring approximately 3 to 3½ cm. at the greatest dimension in the left upper zone. There was no thickening of the hilum. A tentative diagnosis of bronchiogenic carcinoma was made.

Bronchoscopy was done and was negative and a biopsy of the left supraclavicular nodes likewise was negative. On August 20, 1954 a left thoracotomy was done with an incision through the bed of the left fourth rib. The neoplasm was adhered to the third rib and the third intercostal bundle. The third rib was divided on either side of the attachment to the neoplasm and was left attached to the lung and then a left upper lobe lobectomy was done in the usual fashion. Pathologic report was that of alveolar cell type of lung carcinoma with the lymph nodes being negative for metastases.

Postoperatively the patient has done well and a recent check up in February of this year showed no sign of recurrent tumor. It is interesting to note that his clubbing of the fingers has disappeared.

Case 7. This patient was a 63-year-old white female who again had no particular chest symptoms except mild cough. There was no weight loss, hemoptysis, wheezing, etc. A routine x-ray of the chest had shown a density in the lower right lung field and on lateral view this was seen to be a totally solid right middle lobe. Bronchoscopy showed no abnormalities except for lack of air exchange in the right middle lobe. Washings were negative for tumor cells and specific organisms. She was explored on February 9, 1956 with a tentative diagnosis of "middle lobe syndrome." At thoracotomy the upper and lower lobes appeared to be entirely normal whereas the middle lobe was completely solid and airless. A right middle lobe lobectomy was done along with all lymph nodes which could be reached. Pathologic report on this specimen was alveolar cell carcinoma with no evidence of metastasis to the nodes. The patient did well after getting over her initial surgery until June 1956. At this time she began raising small amounts of blood streaked sputum. Physical examination showed rales over the lower right lung field and fluoroscopy and x-ray showed extensive infiltration of the lower half of the right lung field with a large density overlying the anterior end of the fourth rib. It was thought quite certain that the patient had a recurrence of her alveolar cell carcinoma. She was placed on prednisilone and when last seen in the office in August 1956 she had had no further hemoptysis and there was a marked decrease in her sputum. Her weight was stable and x-ray of the chest showed no progression of the disease and it was possible that there was some clearing. She has been lost to follow-up but it is expected that she had had progression of her disease and death by this time.

Case 8. This case was a 58-year-old white male who was seen in consultation on January 8, 1957. He was a known alcoholic and gave a history of marked personality changes developing only about six weeks earlier. These changes consisted of being

uncommunicative and careless in his personal habits. He was admitted to the hospital on January 1, 1957 because of productive cough and fever and when seen on January 8th he had become almost completely non-reactive, incontinent and with rhonchi but no cough or production. X-ray of the chest showed a mass in the posterior portion of the right upper lung field and physical examination showed coarse rales throughout both lung fields. A diagnosis of either lung abscess or bronchiogenic carcinoma with cerebral metastasis was made. Bronchoscopy was done on January 10th at which time a tremendous amount of purulent sputum was seen throughout the tracheobronchial tree with marked redness of the entire tracheobronchial mucosa. The patient exhibited no distress or cough during bronchoscopy in spite of receiving almost no topical anesthesia or preoperative sedation. The bronchial washings were "suspicious" for tumor cells but not definitely so. Monilia was recovered from the sputum. On January 12th the patient developed a flaccid paralysis of his right arm and leg and an inequality of the pupils. On January 22nd the patient suddenly became cyanotic with coarse rales over the entire lung fields. This suggested rupture of an abscess. Emergency bronchoscopy was done without anesthesia and again with removal of several ounces of thick tenacious purulent sputum. A tracheotomy was done at this time and large amounts of purulent sputum, which was very tenacious, was removed at frequent intervals thereafter. The patient remained non-reactive and with the flaccid paralysis in the right arm and leg. Subsequent x-rays of the chest showed no change and he died on February 15, 1957. Postmortem examination showed an abscess in the right frontal area of the brain and in the left side of the cerebellum. A nodule was found in the adrenal. The sections of the lung showed a solid mass in the right upper lobe which contained a large amount of mucin. Pathologic diagnosis was alveolar cell carcinoma involving lung, brain and adrenals. There was no lymph node involvement or involvement of other organs.

Case 9. This case was a 43-year-old white female who was seen because of a cavitary lesion in the left upper lung field. The patient had absolutely no chest symptoms and the lesion was found on a routine chest x-ray made annually because of a history of tuberculosis in her father. There had been some increase in arthritic symptoms over the preceding several months but the patient had had chronic rheumatoid arthritis for many years. Tuberculin was negative and histoplasmin was positive. A tentative diagnosis of histoplasmosis was made and a thoracotomy was done on January 25, 1957. The lesion appeared to be well localized in the upper lobe with no pleural involvement and no lymph node involvement. A left upper lobe lobectomy was done and the resected specimen revealed a cavity. Pathologic report on this was alveolar cell carcinoma. Postoperatively the patient had marked difficulty in getting the lower lobe to expand and eventually a second operation with decortication and partial thoracoplasty was necessary. She had almost immediate marked improvement of her symptoms of arthritis after the operation and at last report she is doing well.

SUMMARY

In alveolar cell carcinoma we have a disease in which the terminology is not completely fixed; there is no agreement as to the etiology or even the location of the tumor. The clinical picture does not present any clear cut pathognomonic signs which lead to the diagnosis prior to surgery in most instances. Therapy has been largely unsuccessful, as in other lung tumors. Occasional five-year survivors occur when early focal disease is resected. This, of course, again speaks for early exploratory thoracotomy for small, asymptomatic densities as seen on routine chest x-ray.

1321 S. 21st Way
Birmingham, Ala.

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SINGLE ADENOMA OF THE THYROID GLAND

D. M. ROYALTY, M.D.

Lexington, Kentucky

It is not feasible nor within the scope of this short paper to cover all the related material regarding single adenoma of the thyroid gland. However, a few of the major controversial issues (with specific reference to papillary carcinoma) will be discussed in an attempt to consolidate our ideas regarding this important entity. Most of these disputed points represent progress in our continuing study of thyroid diseases. In the past decade some of the major controversies appearing in our voluminous thyroid literature are worthy of review for the clinical surgeon. To a great extent the young surgeon, in private practice, must rely on the large clinics and teaching centers as a guide to many of these problems. However, one must be careful in forming an opinion that it is not a mere rearrangement of his prejudices.

Should All Single Adenomas be Surgically Excised?

In 1924 Graham[25] established the relation of malignancy to the single adenoma. Histologic evidence of blood vessel invasion by tumor cells was the criterion for malignant change. Prior to this time such paradoxes as "benign metastasizing goiters" were frequently described in the literature. Continued study has proved that these supposedly benign tumors are malignant in nature. In 1942 King[26] presented evidence to dispute the so-called "lateral aberrant thyroid tumors," and it is universally accepted that these tumors represent metastasis from small papillary carcinomas of the thyroid gland occurring, in nearly all cases, in the homologous lobe.

Except for a few anaplastic thyroid carcinomas, the basis for most of our present knowledge of thyroid malignancies began with the single adenoma. Until recently it was believed by most observers, that approximately 90 per cent of all thyroid carcinomas arise in previously existing adenoma and surgical excision was advocated not only as a definitive procedure but for prevention of carcinoma as well. By recent literature it would seem more likely that the adenoma is actually an encapsulated slow-growing carcinoma from the beginning.

I am not aware of any other field in which the early discovery of a potentially malignant lesion is possible, nor any other lesion more amenable to successful surgical removal with practically no mortality and no morbidity. In addition to this, the size of the adenoma, the age of the patient, and the duration of its presence has no apparent direct bearing on its malignant potential.

In any malignant lesion the best chance of successful surgery is in the interim between development and spread. One who advises that a single adenoma be observed may find that the lesion has metastasized or has spread beyond the capsule. What may have been amenable to total lobectomy now requires more radical surgery. For these reasons it becomes essential to advise the removal of all single adenomas of any size in patients of any age.

In spite of this widely accepted opinion one often finds a dissenting opinion among his internist and physician colleagues, and in a small percentage of patients who refuse surgical therapy for such a small asymptomatic tumor. Recently it has been advocated that use of scintigrams be obtained to determine if the adenoma is single or discrete. The use of I^{131} may determine the activity of these as "warm" or "cold." In patients under the age of forty surgical removal is advised for all "cold" nodules and discrete nodules under 2 cm. in size. In patients between 40 and 60 years of age all single adenomas in men and "cold" ones in females over 2 cm. are removed. In patients past 60 years of age if the adenomas do not have characteristics of malignancy they are observed. Miller and associates[19] have not hesitated to mention the danger of this system of evaluation for general use. However, this is a very interesting study and perhaps indicates some thyroidectomies may be deferred with relative safety.

Does Carcinoma Arise in a Pre-existing Benign Single Adenoma or Is It Malignant from Its Inception?

In the earlier part of this decade the majority of writers were of the opinion that carcinoma of

the thyroid usually arises in a pre-existing benign adenoma. Various percentages are quoted but most agreed that 80 to 90 per cent arise in this manner. Emphasis was placed on the time interval between the discovery of the adenoma and the development of carcinoma. It was admitted by some observers during this time, that it was impossible to substantiate this theory histologically because so often the carcinoma completely replaces the benign adenoma. It was proposed that since some of the carcinomas of the thyroid are slow-growing, that the longer average duration of these tumors suggest they were originally benign.

During the same years an opposing theory contended that there was little possibility that a given adenoma of the thyroid would become malignant, but that the real concern was whether or not the tumor was cancer from its beginning. It was pointed out that one did not see a benign and malignant process occurring in the same adenoma histologically. They are usually homogeneous tumors, each of which is usually all benign or all malignant.

These differences of opinion, a subject for interesting discussion and controversy, has led to very enlightening studies by surgeons and pathologists. Meissner and McManus[20] in discussing this controversy added to the conjecture that many thyroid cancers are malignant from their onset without passing through a benign phase. The evidence that the adenoma transforms from a benign lesion to a malignant one has been mostly clinical. Pathologic demonstration of this actual transformation is not so clear or acceptable. The most striking features in this study, the authors point out, is that in benign tumors a follicular structure is common and papillary structure rare. The exact reverse of this is true in malignant tumors.

In my own practice I have had three cases in which the adenoma was pathologically diagnosed as benign. On serial section a small (2 cm.) focus of papillary carcinoma was found in normal thyroid tissue remote from the presenting lesion. Perhaps, in view of this recent work, clinical surgeons should adopt the philosophy that the discrete tumor of the thyroid is not a benign process which may become malignant but rather a tumor which probably is malignant when first seen. Their surgical attack should include this thought.

The Incidence of Carcinoma in the Single Adenoma

The above widely accepted opinion of surgical removal of all single adenomas is only tenable, if the incidence of carcinoma is sufficiently high. Although the true incidence of carcinoma is unknown, there are numerous reports occurring in the literature on which to base an impression. There is at least one major statistic fallacy in most of the incidence percentage reported, and justly so I might add. This is the difficulty in accurately determining whether or not the adenoma is discrete and to correlate the clinical impression with the pathologic report. It is pointed out that the truly discrete adenoma is uncommon and that what is diagnosed as such clinically often is a prominent adenoma in a multiple nodular gland.

The wide variation of percentages in different locales has suggested to some observers that there may be a geographical incidence of carcinoma. However, as pointed out by Winship,[23] this prevalence may be more closely related to the interest in the disease than in geographic location. It is also apparent that either the incidence of carcinoma is on the increase or some of the multiple nodular glands are mistakenly being included in the statistical reviews. Regardless, it is reasonable to state that the incidence of approximately 25 per cent is sufficiently high to warrant removal of all single adenomas. No one hesitates to suggest removal of a clinically discrete tumor of the breast, for malignancy potential, and yet the reported incidence of malignancy here is under 7 per cent.[12]

Primary Surgical Attack on the Single Adenoma

Through the years there have been many methods of primary surgical attack on the single adenoma. These, briefly, are: (1) Needle biopsy; (2) "nodulectomies" or enucleation; (3) partial lobectomy; (4) bilateral subtotal thyroidectomy; (5) total lobectomy, removal of isthmus and portion of contralateral lobe; (6) total thyroidectomy.

Whether one advises surgical removal of a single adenoma for prophylaxis of carcinoma or his belief that it is a carcinoma from its inception, it is apparent to this observer that the same diligence in management should be employed as is the usual custom in malignant or potentially malignant neoplasms elsewhere. I

agree with Klopp[15] that until papillary carcinoma has proved to have a different biologic behavior from that of other cancers, it should not be surgically handled in any different fashion. An established feature of papillary carcinoma is its ability to implant itself in wounds and metastasize locally when avenues of vascular spaces are invaded either by the normal evolution of growth or by growth from inadequate local operation. Crile,[8] among others, has reported a high percentage of residual tumor in the remnant of the lobe which had been previously operated on for single adenoma and found to have carcinoma. It has also been pointed out that the poorest over-all results are in those cases of incomplete local excision of the primary tumor and approximately 30 per cent of these cases develop metastases.

It is my opinion that total lobectomy with removal of the isthmus and portion of the opposite lobe should be the primary surgical attack for a single adenoma for the following reasons:

1. This procedure allows for complete removal of the tumor with less chance of seeding or spreading.

2. It avoids the necessity of re-entering a field which has recently been explored surgically if the pathologist, unable to make a positive diagnosis on frozen section, finds invasive carcinoma on routine serial section.

3. Surgical results, over-all, are excellent as proven by the literature.

4. It is technically less difficult than a lesser procedure.

Total thyroidectomy meets those requirements that I have outlined above and has many advocates according to the literature. Time may prove this to be the more plausible approach; however, most are in agreement with total lobectomy.

Extent of Surgery

What constitutes reliable critera for malignancy in a single adenoma and the extent of the surgery which should be undertaken is controversial.

It is evident that frozen section diagnosis of a single adenoma may be as difficult as any problem facing the pathologist. Correlation of the surgical understanding of the process and its future evolution with the pathologic study becomes necessary in each individual case. Contrary to the early belief, blood vessel invasion alone is not an indication for radical surgery. There is apparent agreement that certain findings constitute requirement for radical surgery:

1. The histologic identification of thyroid tissue in a cervical node. So-called "lateral aberrant thyroid."

2. A diagnosis of cancer with palpable regional node metastases.

3. Invasion of the capsule of the adenoma by carcinoma.

4. Any tumor which is diagnosed as grade II or higher.

If frozen section is not diagnostic one should wait for permanent sections before deciding on the extent of the operative procedure.

There is marked difference of opinion in the literature as to the extent of neck dissection which should be undertaken for papillary carcinoma of the thyroid. There are those who contend that this problem should have special consideration because of its individual behavior pattern. They point out the long evolution of many patients regardless of type of treatment. Many of this group believe that spread is only to the cervical lymph nodes and distant metastases is rarely or never seen. They advocate a limited neck dissection with preservation of the sternocleidomastoid and the jugular vein, thereby removing the grossly involved nodes.

Another theory to which I adhere, is that, although the cancer is slow-growing it must be treated as any malignancy and that because of its behavior pattern one has a better chance at successful treatment. The studies indicate that most papillary tumors have a follicular component and therefore are capable of distant metastases. Recent reports[10] in the literature indicate that many papillary cancers are invasive, do metastasize distally and are difficult to eradicate. The same principle of block dissection as employed elsewhere reduces the chances of leaving involved nodes which would require removal at a later date.

Therefore the best opportunity to remove all the cervical nodes and possibility of recurrent or distant metastases is by radical neck dissection. The pathologic criterion of malignancy of nodal metastases, capsular invasion, and grade II or higher is indication for radical neck dissection. If carcinoma is found in the contralateral lobe it

is advisable to do bilateral neck dissections with an interval of time between surgical procedures. There are no indications for prophylactic neck dissections.

SUMMARY

1. All single adenomas should be surgically excised except when surgery becomes a greater risk than the disease operated for.

2. The single adenoma is not a benign process which may become malignant but is probably malignant from its inception.

3. The percentage of carcinoma occurring in excised single adenomas justifies their removal.

4. Total excision of involved lobe, isthmus and portion of contralateral lobe as the primary surgical attack should be adopted.

5. Radical neck dissection should be carried out on the involved side when criteria for invasive malignancy is established.

152 E. Maxwell St.
Lexington, Kentucky

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DELAY IN THE DIAGNOSIS OF BREAST CARCINOMA

GEORGE P. ROSEMOND, M.D., F.A.C.S.

Philadelphia, Pennsylvania

"Few as are the complete recoveries from cancer of the breast after operation, I believe they will grow more frequent when the family doctor, to whom they are generally first shown, urges operation earlier, for at present they usually let the best time for operation slip by, and the women do not consult professed surgeons until the local disease and the affection of the axillary glands are so far advanced that a complete operation is no longer practicable."

This paragraph is a direct quote from Billroth's *Surgical Pathology* which, as revised and translated into English in the year 1889, was said to represent the most advanced surgical thinking up to that time.

About this same time, William S. Halsted began to clean out the axilla routinely. When Dr. Halsted reported the results of radical operations for the cure of cancer of the breast at The Johns Hopkins Hospital, the five-year survival rate was 28.9 per cent with a relative cure rate of twenty-four per cent.

I am not here to discuss treatment of breast cancer but many feel that Halsted's original radical mastectomy has not been appreciably improved upon and that the present somewhat more optimistic statistics are due to other factors.

Prognosis in breast cancer has been said to be influenced by two sets of factors: (1) Uncontrollable factors, such as sex, age, heredity, etc.; (2) Controllable factors, listed as (a) delay in treatment and (b) type of treatment.

As for the type of treatment, advocates of radical mastectomy have proven conclusively that this form of treatment offers the best prognosis as evidenced by numerous publications from Halsted to Haagensen and *ad infinitum*. Urban is in the process of proving that extended radical mastectomy is the only true answer, and McWhirter has shown that radical mastectomy is superfluous and simple mastectomy plus x-ray therapy is the only sensible approach. Recently, one investigator, Rosahn[6] has demonstrated that it makes little difference how cancer of the breast is treated. Unfortunately, as far as I know, no crusader urging departure from the generally accepted radical mastectomy has attempted randomization which might prove or disprove his theory.

Even the question of delay in diagnosis has aroused some controversy. McKinnon[11] has suggested that delay is unimportant, affecting prognosis but little. The variable life history of breast cancer seems to support "illogically" logical theories. Keyes and associates have presented some interesting although somewhat indefinite data comparing the longevity of 100 treated and 100 untreated cases. Although one might question on several counts the exact accuracy of their statistics, it seems evident that radical mastectomy definitely prolongs the life of the breast cancer patient. They indicate that the life span is doubled by treatment. Certainly, Daland's 22 per cent five-year survival in untreated cases is far below what we should expect, and most of this 22 per cent, although living, must have been very unhappy as they approached the five-year mark.

The Cancer Control Committee of the Philadelphia County Medical Society believes that early diagnosis is important and, to a certain extent, controllable. In 1951, responding to an urge for critical self-analysis, it established a committee for the study of delay in the diagnosis of breast cancer. This work is supported by the Philadelphia Division of the American Cancer Society. The ultimate hope is to establish a cooperative city-wide registry which will correlate early diagnosis and prognosis. Data concerning other factors, too numerous and so far too nebulous to mention here, will inevitably appear. Although we are still very much in the formative period of our development, certain data seem worthy of scrutiny.

Since our committee went to work, the records of 3343 patients have been evaluated. In most

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instances, the patients were interviewed. Critical retrospective analysis indicates that, although physician delay is on the decline in our area, we still have a definite and real job to do. Last year, we surveyed 864 cases. Physician delay figures were under 25 per cent for the first time.

To focus emphasis, it seems proper to call attention to certain factors.

Table 1 points up the fact that the first sign noted in the great majority of patients is a lump in the breast. Other initial signs and symptoms follow as listed in table 1.

Table 2 points out clearly who discovered the sign or symptom indicating the cancer. In most instances, the lesion was first noted by chance. If the gospel of routine breast self-examination could be convincing enough without producing universal psychoneurosis, the cause of early diagnosis would be advanced considerably. In recent years, our Philadelphia physicians are showing signs of improvement. This 8 per cent figure was once four per cent.

TABLE 1

*Initial signs and symptoms of breast carcinoma**

First Symptom Noted	No.	Per cent
Mass in breast	1402	88
Bleeding or discharge from nipple	43	3
Inverted nipple	32	2
Mass in axilla	32	2
Pain	26	2
Dimpling skin	10	1
Ulcer	8	1
Swollen arm	2	—
Uncertain	20	1

* 1575 cases.

TABLE 2

*Discovery of initial signs and symptoms of breast carcinoma**

Discovered by:	No.	Per cent
Patient (accidentally)	1405	88
Patient (purposeful self-examination)	67	4
Physician	170	8

* 1575 cases.

TABLE 3

*Incidence of breast carcinoma in the male and female**

	No.	Per cent
Female	1566	99.4
Male	9	0.6

* 1575 cases.

TABLE 4

*Age at time of interview of patients with breast carcinoma**
2336 cases

	Years
Youngest	21
Oldest	97
Average	55

* Only one patient was under 25 years of age.

Table 3 merely points out again the rarity of cancer of the breast in the male.

Table 4 points out that of the last 2336 cases, the average age at the time of the interview was 55 years. The oldest patient was 97 years of age, illustrating the association of this tumor with advancing years. Of special interest is the fact that only one patient in the entire group was less than 25 years of age. This indicates clearly that we should save our propaganda ammunition for the mature woman. Also, we should avoid psychic trauma in the young woman and disfiguring biopsy in the adolescent since cancer of the breast in either of these two groups is almost a medical curiosity. I do not know of a proven case of breast carcinoma in the undeveloped, prepuberal female breast. Still, deforming biopsy for mastitis of puberty has been suggested by the uninformed.

Early diagnosis of breast cancer can be facilitated by proper utilization of diagnostic measures followed by prompt biopsy. Careful evaluation of the female breast on physical examination should be repeated every six months in the 35-year-old-plus female. The experienced examiner can usually accurately evaluate the breast. It is well to enlist the help of the mentally stable woman by coaching her in breast self-examination to be carried out after each menstrual period.

Needle aspiration is extremely helpful in differentiating cystic and solid masses. Cystic

masses which can be eliminated permanently by aspiration are not significant. The incidence of cancer occurring in a cyst wall is less than five-tenths per cent. Adequate follow-up will pick up this very small number reasonably early. Successful aspiration is often a safe substitute for surgery.

The place of x-ray of the breast as a diagnostic tool has not been definitely established. I would like to quote a recent personal communication from Dr. Jacob Gershon-Cohen who is the principal advocate of this method.

"In our survey group of 1300 *asymptomatic* women during the past two years, ten cases of cancer have come to operation. Of these, six patients had no palpable tumor and the surgeon had to be prevailed upon to carry out diagnostic resections. In the other four cases, masses were palpable, but none of these was dominant, and again, surgery was done solely because of the x-ray findings."

Unfortunately, few radiologists have shown the enthusiasm of Dr. Gershon-Cohen and the method has not won widespread acceptance.

May I warn against over zealous efforts at biopsy. One friend of mine, disturbed by the parade of delay cases appearing before our Philadelphia Committee month after month, has suggested a five-fold increase in biopsy and mentioned a ten or twelve to one ratio of benign to malignant disease at biopsy as acceptable. Such ideas could promote hysteria in some and dreams of easy financial returns in others. We should advocate biopsy when there is an identifiable lesion to biopsy, frequent observation when in doubt, and recheck every six months for the female past 35 years of age with the so-called normal breast.

If I may be allowed to wander slightly from my topic, I would like to speculate that much of the confusion and bias relating to the diagnosis and treatment of breast cancer could be eliminated if large groups of investigators would coordinate and attempt to standardize methods of evaluating obtainable data, and if each person who strays afield from accepted methods would introduce randomization into his project.

SUMMARY

The prognosis of breast cancer can be improved by early diagnosis. Philadelphia County has established a self-critical committee to study the problem and to analyze obtainable data.

The lesion suggesting breast cancer is usually first noted by the patient and most often manifests itself as a lump.

Breast cancer is so obviously a blight of advancing years that it seems unnecessary and possibly even psychically harmful to propagandize it to the youthful.

Early diagnosis can be facilitated by an awareness of the early manifestations of carcinoma by promptly establishing the suspected diagnosis by biopsy. The aid of the patient should be solicited to assure universal early diagnosis.

Aspiration of breast masses to differentiate cystic from solid tumors is useful.

The place of x-ray diagnosis has not been established although at least one preliminary report offers some promise.

Temple University Medical Center
Philadelphia, Pa.

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ESOPHAGO-DIVERTICULOSTOMY FOR STENOSIS OF UPPER ESOPHAGUS ASSOCIATED WITH ZENKER'S DIVERTICULUM*

EDWARD F. SKINNER, M.D., AND GENE PAGE, M.D.

Memphis, Tennessee

The following case report gives a simple solution to a troublesome problem. This elderly patient had an almost complete stenosis of the upper esophagus associated with a Zenker's diverticulum of the esophagus. Our solution to the problem was to leave the stenosed esophagus in place and simply short circuit food around it by anastomosing the diverticulum itself to the esophagus just below the stricture. This was carried out with a very nice clinical result.

A diligent search of the literature has revealed no similar case treated in this manner.

It is possible that such an operation could be useful occasionally for a traction diverticulum in the mid- or lower esophagus associated with stenosis of the esophagus.

Mosher[6] attempted endoscopically to open the wall between the esophagus and the diverticulum producing a diverticulo-esophageal anastomosis. However, a prohibitive mortality rate resulted because of infection and mediastinitis.

Mortenson and associates[5] advocated a one-stage procedure for the treatment of esophageal diverticula. The mortality and morbidity rates were comparable with the two-stage procedure, the period of hospitalization shorter, and only one operation and anesthetic was necessary.

Lahey and Warren[3] championed the two-stage procedure. According to their beliefs a distal obstruction is necessary in order to produce a diverticulum. They classified diverticula into early, moderate and more advanced stages and the patient reported in our article would fall into their third-stage diverticulum classification. According to Lahey and his group in these third-stage type diverticula the swallowing mechanism carried food directly into the diverticulum rather than into the normal channel of the esophagus, and the food enters the esophagus itself only in a spill-over fashion. This is probably the explanation for the excellent swallowing that resulted in our patient after a direct anastomosis between the large diverticulum and the esophagus below the stenosis.

CASE REPORT

Mr. B. K. C., a white male, aged 81 years, was referred to us by Dr. C. M. Murry of Oxford, Mississippi, who found a severe stenosis present in the upper esophagus on November 14, 1957 and was unable to pass an esophagoscope beyond this point. The patient was admitted to the Baptist Memorial Hospital in Memphis on November 15, 1957; his chief complaint was inability to swallow food or even water for several days. He stated that he had had difficulty in swallowing for a period of about two months prior to admission and had occasional regurgitation for several years. No pain was associated with the symptoms.

The patient gave a history of having suffered a coronary occlusion at the age of 76 but apparently this was not a severe attack and he made an uneventful recovery in a period of about two months. He stated that he noted some shortness of breath which was noticeable after walking about one-half block. He had no pain-effort syndrome but described an uncomfortable feeling in the upper substernal region attributed to indigestion. He had never taken nitroglycerine or digitalis. He became quite depressed following the recent death of his wife and lost most of his zest for living. His health, other than described above, had always been good except for an impairment in hearing.

He stated that he carried out a little light work in the yard. He had a slight cough productive of white scanty sputum with hard lumps in it. He gave no history of hemoptysis. Slight edema of the left ankle had been noted occasionally. He smoked two or three cigarettes daily. He was taking no medication prior to his admission. There had been no fever, chills or night sweats and he gave no history of allergy. He stated that he had some weight loss prior to admission of an undetermined amount. He had noted fatigue on mild exertion.

He had a past history of pneumonia twenty years previously and a prostatectomy seven years ago. About ten or fifteen years ago he fell in the bathtub and fractured several ribs.

Physical examination revealed a well preserved man for the stated age of 81 years. He appeared quite comfortable with a pulse rate of 70 per

* From the Department of Surgery, University of Tennessee School of Medicine and the Baptist Memorial Hospital, Memphis, Tennessee.

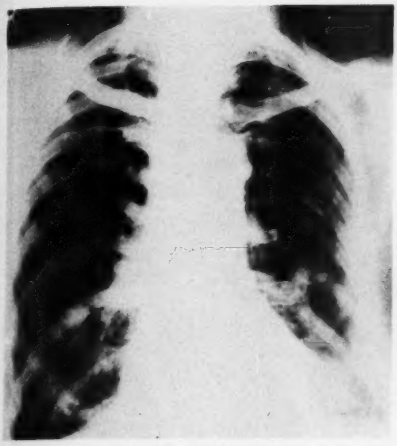

FIG. 1. A PA chest film made November 16, 1957 shows considerable deformity of the left chest caused by old fractures of ribs which have healed with good callus formation. The heart is slightly enlarged and shifted to the left. Both leaves of the diaphragm are flattened and depressed, particularly on the left. In the right lung there is a 2.5-cm. granuloma near the base with a dense central calcification. The lung otherwise is relatively clear. No active parenchymal lesions are seen in the left lung but there is a suggestion of emphysema throughout, particularly in the apex.

minute with a number of premature contractions. The blood pressure was 140/80, the heart was enlarged to the left, with the apex in the fifth intercostal space about midway between the midclavicular and anterior axillary line. The first heart sound at the apex was replaced by a Grade II systolic murmur which extended throughout systole. The sounds at the aortic and pulmonic areas were essentially normal and the lungs were clear to physical examination; there was some decrease in breath sounds bilaterally. The abdomen was normal with the liver, spleen and kidneys not being palpable. There was no edema of the extremities on admission. He was somewhat hard of hearing and the skin was flabby and dry. The remainder of the physical findings were negative. His routine laboratory studies were relatively normal. The total blood protein was 5.5 gm. with albumin 3.8 gm. and globulin 1.7 gm., with an A-G ratio of 2.21.

An electrocardiogram revealed a sinus rhythm with a rate of 70 per minute with many ventricular premature contractions all originating from the same focus. There were a number of auricular premature beats. The changes in leads II, III and AVF were compatible with an old posterior infarction according to the consulting cardiologist, Dr. Lawrence L. Sebulsky. The T-waves were of low voltage. Doctor Sebulsky concluded that the patient had arteriosclerotic heart disease with a remote infarction and was fairly well compensated. In view of the many premature contractions and shortness of breath related to effort, he was digitalized by hypo prior to surgery (the patient was unable to swallow pills).

Barium studies were done and revealed a moderate sized Zenker's pharyngo-esophageal diverticulum and almost complete stenosis of the upper esophagus. Chest films showed multiple healed rib fractures on the left and considerable pulmonary fibrosis. There was a small granulomatous nodule in the right middle lobe, possibly either a histoplasmoma or a tuberculoma. The heart was considerably enlarged and there was calcification in the arch of the aorta.

OPERATION

On November 19, 1957, an esophago-diverticulostomy and esophagoscopy was performed by the authors assisted by Dr. John R. Hall, under

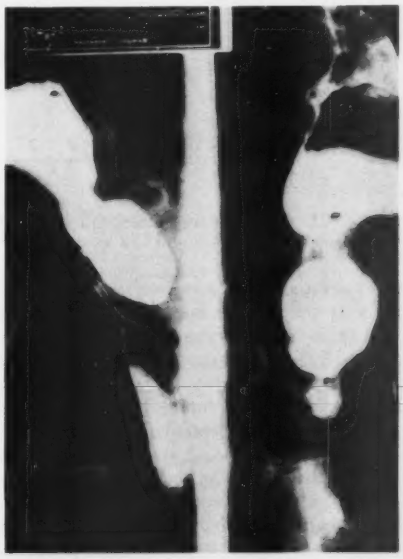

FIG. 2. PA and oblique projections of the pharynx and upper esophageal area outlined by barium (made on November 16, 1957). The diverticulum measures about 3.5 cm. in diameter and appears as a rounded shadow just below the pharynx, directly in the midline. The oblique projection shows the diverticulum posterior to the stenotic portion of the upper esophagus and there is approximately a 1-cm. gap between the lower tip of the diverticulum and the esophagus just below its stenosed portion. It was this gap which was bridged at surgery by an esophago-diverticulostomy.

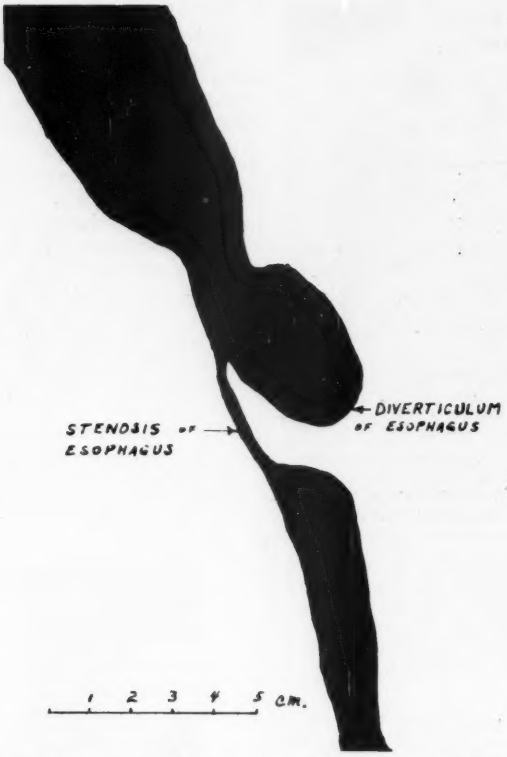

FIG. 3. This is a positive direct tracing of figure 2 to illustrate the size of the gap to be bridged between the diverticulum and the normal esophagus just below the stenosis.

general anesthesia. A 10-cm. vertical incision was made over the anterior border of the left sternomastoid muscle and by sharp and blunt dissection the thyroid gland was exposed. The middle thyroid vein and inferior thyroid artery on the left were doubly ligated and divided. The esophagus was exposed; there was a diverticulum off the esophagus posteriorly measuring approximately 3.5 cm. in size and just below the neck of the diverticulum there was a marked stenosis of the esophagus. The esophagus was exposed below the stenosis and a Penrose drain placed around it to elevate it for inspection of the entire esophagus in this area. After these structures were thoroughly exposed, Dr. Hall passed an esophagoscope into the upper esophagus and was unable to pass it down through the strictured area even under direct guidance of the surgeons working in the neck. Even dilators would not pass through the stenosed area, including the small #11 Jackson esophageal dilator. The esophagoscope was removed and an anastomosis was then made between the tip of the diverticulum and the infra-stenotic portion of the esophagus just above the sternum. A two-layer anastomosis was made using #000 silk as interrupted stitches, reinforced with mattress stitches of #000 chromic catgut. The wall of the diverticulum was approximately 3 mm. thick and it was possible to get a very good closure of the anastomosis. When the anastomosis was almost complete a Levin tube was passed through the anastomosed area into the stomach which was later connected to constant Wangensteen suction. A Penrose drain was placed in the lower corner of the incision and the wound was closed loosely in layers using running stitches of #000 chromic catgut for the strap muscles and subcutaneous tissues and a running stitch of #00 Deknatel for the skin. The postoperative condition was good. There was no evidence of tumor seen either through the esophagoscope or in the neck.

Two days later the Levin tube and drain were removed and the patient was started on a clear liquid diet. His digitalis was continued and he

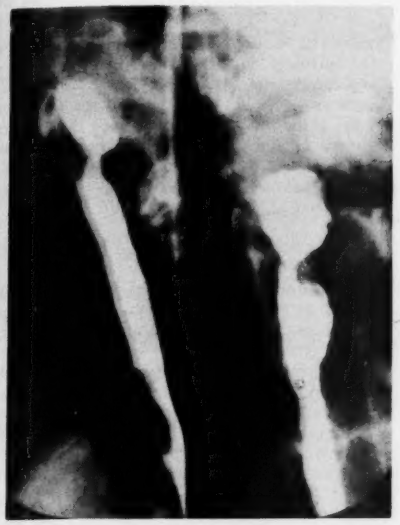

FIG. 4. PA and oblique projections of the pharynx and upper esophageal area as outlined by barium on February 5, 1958. There is now an adequate lumen between the diverticulum and the esophagus (below this area) with rapid passage of the barium through the area. In addition the stenosed portion of the esophagus is still present and outlined by a thin trickle of barium.

made a very fine recovery clinically. The incision healed by primary intention and his stitches were removed a week following surgery. He was discharged from the hospital on November 27, 1957, on his eighth postoperative day, with instructions to follow a regular diet, but to grind all his meat for a period of two months. The premature cardiac contractions persisted but to a lesser degree than before surgery. He had some soft tissue swelling of the feet which was non-pitting, and the digitalis was to be continued at home under the supervision of his family physician, Dr. Hugh Boswell of New Albany, Mississippi.

The final diagnosis was a Zenker's pulsion (pharyngo-esophageal) diverticulum with esophageal stricture, benign; arteriosclerotic heart disease and remote coronary infarction; pulmonary granuloma, inactive.

At the time of discharge the patient had no dysphagia; he was eating a regular diet with ground meat. He was afebrile after the first postoperative day. He was ambulatory from the second postoperative day, and had surprisingly little discomfort in the area of the incision.

The patient was in our office on February 5, 1958 for a check-up examination at which time his general condition appeared excellent.

FIG. 5. This is a positive direct tracing of figure 4 illustrating the postoperative result with barium traversing this area in two separate lumens.

A new chest film showed no change when compared with his preoperative films. The granulomatous lesion in the right lung had not changed in size.

The patient was eating a regular diet although his meat was still being ground and he was taking Alcaroid, one teaspoonful in water, after every meal containing meat. He still had hiccoughs at times; also he stated that he had some burping after eating and some difficulty in swallowing, but no pain.

Repeated esophagograms were made and are reproduced herewith. The thick barium paste traversed the new opening between the diverticulum and lower esophagus rapidly and a thin trickle of barium still goes down the stenosed portion of esophagus. The patient was advised to continue on his present diet and medication indefinitely, and to have periodic check-up examinations. Again there was no evidence of neoplasm.

SUMMARY

The case is presented of an elderly white male patient with a severe stenosis of the upper esophagus and an associated Zenker's diverticulum.

A simple solution to the problem was a direct anastomosis between the diverticulum and the normal esophagus below the stenosis, which was performed as a one-stage procedure with a very nice clinical result. The stenosed portion of esophagus was left in place without resecting it, because of the patient's advanced age and associated heart disease. An end-to-side anastomosis was utilized for the operation.

It is conceivable that this same procedure might be occasionally useful in strictures of the mid-esophagus associated with a traction type diverticulum.

899 Madison Ave.
Memphis, Tenn.
(Dr. Skinner)

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STERILITY OF MULTIPLE DOSE VIALS AFTER REPEATED USE*

JACK A. YOUNG, M.D., THOMAS S. COLLETTE, B.A., AND WILLIAM F. BREHM, M.D.

Sayre, Pennsylvania

During the course of clinical anesthesia many agents are used from multiple dose containers. It is fairly common practice to attempt sterilization of the rubber stopper by wiping it with an antiseptic solution before use. Needles of doubtful sterility from being exposed to room air are then plunged through the questionably sterile rubber stopper to aspirate the solution. From time to time the question had crossed our minds as to the sterility of the last cc. or so of medicament remaining in the vial. The question became more insistent when solutions of succinylcholine were considered. At the end of an operating day as much as half of a 100-, 250- or 500-cc. bottle of this solution would be left. From several operating rooms there might be a considerable quantity of possibly contaminated drug remaining by nightfall. Should this be discarded or could it be safely used another day?

As a precautionary measure we had been discarding all multiple dose vials as soon as they had been reduced to a remaining 1, 2 or 3 cc., except for our solutions of succinylcholine. We had been making our dilute solutions of succinylcholine in sterile saline so that there would be no nutrient for bacterial growth. Any of this solution remaining at the end of the operating day was refrigerated for re-use the next day, but any more than 72 hours old was discarded. We made our own high-potency succinylcholine by the following method. By discarding 20 cc. from a 100-cc. vial of sterile distilled water and adding 2,000 mg. of succinylcholine in 20-cc. volumes, we made 100-cc. bottles of 2 per cent solution. These bottles were always refrigerated when not in use. To avoid the use of contaminated solutions we discarded the last 4 or 5 cc. of solution. By following this pattern, we had avoided any definite reactions due to questionably sterile drugs.

However, we had begun to question the use of succinylcholine in saline for operative patients, since surgical patients usually require little or no salt intake for 24 to 72 hours postoperatively. It seemed irrational to give some patients as much as 1,000 cc. of isotonic saline *via* a succinylcholine drip. At this point, we changed to 5 per cent glucose and water as the vehicle. To be sure of the sterility of this solution formula, we made spot bacteriologic cultures of these succinylcholine solutions. During a year and a half, these spot checks had been negative for bacterial growth.

All of our precautions and the negative spot checks did not allay our uneasiness when we handled nearly exhausted ampules. A search of the literature back to 1921 revealed nothing specific regarding this possible source of contamination. Post[1, 2, 3] and Moor[4] had done some nice work on sterilization of ophthalmic solutions and instruments but these studies yielded no specific answers to our questions.

We accordingly decided to study the survival time of bacteria in various solutions used in clinical anesthesia.

THE STUDY

Part one. The first part of the study consisted of a random spot check, during a three-month period, of cultures made from a variety of solutions from clinically used containers. Two cc. of each agent were removed from each solution and cultured in thioglycollate broth. These broths were incubated at 37°C. for four days. In all cases the cultured broths remained sterile, showing the solutions to be safe for use. Table 1 indicates the variety of agents, the number of cultures and the results of this spot check.

Part two. In the second part of the study, equal amounts of 10 selected solutions were inoculated with a known quantity of bacteria to check bacterial survival time in the solution.

With sterile syringes, 9.9 cc. of each of the 10 test solutions were transferred to sterile screw-capped tubes. Two controls were set up in similar fashion using physiologic saline. To each of these tubes was added 0.1 cc. of a saline suspension of

* From the Departments of Anesthesiology and Bacteriology, Guthrie Clinic-Robert Packer Hospital, Sayre, Pennsylvania.

TABLE 1

Solutions checked for sterility

Solution	Number Cultured	Results
Anectine chloride	8	No growth
Dextrose 5%	3	No growth
Dextrose 5% with Anectine chloride	11	No growth
Flaxedil	3	No growth
Isotonic sodium chloride with Anectine 0.1%	2	No growth
Isotonic sodium chloride with Anectine 2.0%	1	No growth
Istotonic sodium chloride	1	No growth
Parenteral water	3	No growth
Pentothal 5%	3	No growth
Pontocaine hydrochloride 0.15%	4	No growth
Sympocaine* solution 0.5%	1	No growth
Syncurine	2	No growth
d-Tubocurarine chloride	3	No growth
Xylocaine hydrochloride 1%	2	No growth
Xylocaine hydrochloride 2%	3	No growth
Xylocaine hydrochloride 2% with 1:100,000 epinephrine	2	No growth
Total number of spot cultures	52	

* Winthrop Laboratories (experimental).

an 18-hour culture of a laboratory strain of *Micrococcus pyogenes* var. *aureus*. This suspension was estimated to contain 10,000 organisms per cc. To serve as an initial control count, 1 cc. was immediately transferred from the saline controls to sterile Petri dishes and pour plates made with nutrient agar. The inoculated solutions were left at room temperature and after a period of 30 minutes, 1 cc. was transferred from each solution to a sterile Petri dish. Pour plates were made in the usual manner with nutrient agar. This procedure was repeated after two hours had elapsed, and after one, two, and three days. All cultures were incubated for 48 hours at 37°C. and bacterial counts were made. These data are summarized in table 2. Survival percentages were calculated using the averages of the two initial control counts as the numerator, and the bacterial counts from the various solutions as the denominators. Survival percentages in the case of the controls were calculated using the actual count figure for that particular control as the denominator.

This procedure was then repeated using an inoculum estimated at 100,000 bacteria per cc. These data are summarized in table 3. Following the evaluation of these figures, as given in tables 2 and 3, it was concluded that the test organisms were able to survive for the longest period of time in Pontocaine hydrochloride.

TABLE 2

*Bacterial counts and survivor percentages**

Solution	pH	Survival Time								
		30 Minutes		2 Hours		1st Day		2nd Day		3rd Day
		Count	*Per cent*	*Count*	*Per cent*	*Count*	*Per cent*	*Count*	*Per cent*	*Count*
Pontocaine hydrochloride 0.15%	5.25	66	75	70	79.5	6	6.8	1	1.1	0
Nembutal sodium 50 mg./cc.	10.45	1	1.1	0		0		0		0
Xylocaine hydrochloride 1%	6.7	47	53.4	13	14.7	0		0		0
Xylocaine hydrochloride 2% with epinephrine	3.4	1	1.1	0		0		0		0
Xylocaine hydrochloride 2%	6.75	11	12.5	0		0		0		0
Atropine sulfate 0.4 mg./cc.	3.9	36	40.9	0		0		0		0
Tubocurarine chloride 3 mg./cc.	3.53	68	77.2	33	37.5	0		0		0
Syncurine 1 mg./cc.	6.5	44	50	1	1.1	0		0		0
Flaxedil 20 mg./cc.	2.8	61	69.3	36	40.9	1	1.1	1	1.1	0
Pentothal 5%	10.0	48	54.5	10	11.3	2	2.2	0		0
	Initial Count									
Saline control A	90	84	93.3	76	84.4	3	3.3	2	2.2	0
Saline control B	86	81	94.1	73	84.8	1	1.1	0		0

* *Micrococcus pyogenes* var. *aureus*, 10,000/cc.

TABLE 3

*Bacterial counts and survivor percentages**

Solution	pH	Survival Time								
		30 Minutes		2 Hours		1st Day		2nd Day		3rd Day
		Count	*Per cent*	*Count*	*Per cent*	*Count*	*Per cent*	*Count*	*Per cent*	*Count*
Pontocaine hydrochloride 0.15%	5.25	750	98.6	620	81.5	42	5.5	0		0
Nembutal sodium 50 mg./cc.	10.45	0		0		0		0		0
Xylocaine hydrochloride 1%	6.7	580	76.3	158	20.7	0		0		0
Xylocaine hydrochloride 2% with epinephrine	3.4	50	6.5	1	0.1	0		0		0
Xylocaine hydrochloride 2%	6.75	130	17.1	45	5.9	0		0		0
Atropine sulfate 0.4 mg./cc.	3.9	248	32.6	0		0		0		0
Tubocurarine chloride 3 mg./cc.	3.53	586	77.1	87	11.4	0		0		0
Syncurine 1 mg./cc.	6.5	342	45.0	1	0.1	0		0		0
Flaxedil 20 mg./cc.	2.8	454	59.7	247	32.5	0		0		0
Pentothal 5%	10.0	388	51.0	82	10.7	0		0		0
	Initial Count									
Saline control A	768	730	95.0	622	80.9	36	4.1	14	1.8	0
Saline control B	752	714	94.9	562	74.7	20	2.6	4	0.5	0

* *Micrococcus pyogenes* var. *aureus*, 100,000/cc.

TABLE 4

*Bacterial counts and survivor percentages**

Solution	pH	Survival Time								
		30 Minutes		2 Hours		1st Day		2nd Day		3rd Day
		Count	*Per cent*	*Count*	*Per cent*	*Count*	*Per cent*	*Count*	*Per cent*	*Count*
Pontocaine hydrochloride 0.15%	5.25	76	69.7	71	65.1	10	9.1	4	3.6	0
Pontocaine hydrochloride 0.15% with epinephrine 1:200,000	6.25	16	14.6	12	11.0	1	0.9	0		0
	Initial Count									
Saline control A	112	105	93.7	92	82.1	8	7.1	3	2.6	0
Saline control B	106	99	93.3	94	88.6	6	5.6	1	0.9	0

* *Micrococcus pyogenes* var. *aureus* with and without epinephrine added to Pontocaine.

To check these results, another run was made using Pontocaine hydrochloride both with and without epinephrine as the test solutions. Survival time was again longer than the average in the other solutions, but a more abrupt falling off in the bacterial population was noted in the solution containing epinephrine. These data are listed in table 4.

DISCUSSION

The above procedures were run in a busy clinical laboratory and, of necessity, the techniques and controls used were not elaborate. However, it seems evident that certain conclusions may be drawn from the data as presented: (1) none of the solutions tested acted as a nutrient medium for the bacteria; (2) there was no increase in the bacterial population but rather a decline, occasionally quite abrupt; (3) most of the solutions tested appeared to have a lethal effect on the test organisms. All solutions but Pontocaine showed a markedly more rapid decline of population than the saline controls.

The number of bacteria used for inoculation of these solutions was considerably larger than any conceivable accidental inoculation which might

occur during the actual use of these vials. In spite of the relatively large number of bacteria introduced, a considerable proportion of the vials had sterilized themselves after two hours, the majority were sterile after the first day and all, including the saline controls, were sterile after three days. It would appear from the above data that many of the solutions in use in the operating room are self-sterilizing to various degrees. At one extreme in the solutions tested would be Nembutal sodium, apparently very lethal for bacteria, and at the other extreme would be Pontocaine hydrochloride in which the bacterial population declined roughly parallel with the population of the saline controls.

No attempt was made to evaluate each solution for the factors which made it bactericidal. The pH was determined for each but from the results this was not a major factor in the bactericidal effects of the various solutions (see tables 3 and 4).

It seems evident that accidental contamination of vials which are punctured a number of times may occur but that the self-sterilizing properties of these solutions is a prime factor in preventing infections from this source.

It would appear that more caution should be exercised before repeated use of the same bottle of Pontocaine. The addition of epinephrine markedly enhances its bactericidal effects and would probably be a good idea from the point of view of both sterility and anesthesia. However, one should temper his actions by the knowledge that Pontocaine is practically sterile by the second day after gross contamination, and that all of our spot cultures of clinically used vials of Pontocaine and other medicaments were sterile.

SUMMARY

Two groups of tests were made to determine the sterility or contamination of selected drugs in multiple dose containers used in clinical anesthesia.

Test one comprised a spot check culture of 52 solutions selected at random from partially used containers during a three-month period.

Test two consisted of bacterial counts made at 30 minutes, two hours and one-, two- and three-day intervals on 10 selected agents which had been inoculated with controlled numbers of *Micrococcus pyogenes* var. *aureus*.

CONCLUSIONS

Spot checks of clinically used multiple dose vials showed no contamination.

Deliberate gross contamination of 10 selected agents showed them to be, with one exception, practically sterile by the end of one day.

Pontocaine hydrochloride 0.15 per cent was the slowest to sterilize itself although it was finally sterile after two or three days.

The addition of epinephrine to Pontocaine hydrochloride increased the sterilization process and brought this agent within the acceptable limits of performance.

The pH of the agents did not, apparently, influence their self-sterilizing capacity.

There appears to be a fairly rapid self-sterilizing activity in all of the 10 selected agents used in this study.

Guthrie Clinic-Robert Packer Hospital
Sayre, Pa.

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MECKEL'S DIVERTICULUM

ELTON S. THOMAS, M.D.

Columbus, Mississippi

Since 1812, when Meckel first described this anomaly, the incomplete obliteration of the omphalomesenteric duct has been the subject of much interest. It is, however, still considered an infrequent finding at the operating table and very few surgeons have compiled a large series of personal cases. Attention was focused on this congenital anomaly when the first nine cases of this series were encountered in a relatively short period of time. Fifteen cases were encountered during a four-year period, from January 1, 1954, to December 31, 1957.

INCIDENCE

The surgery was performed at two separate fifty-bed hospitals, and 705 laparotomies were done. Thus, the incidence of Meckel's diverticulum in this series is 2.1 per cent. This parallels the incidence which has been recorded in numerous autopsy series, namely 2 to 4 per cent, but it is much higher than the frequency usually reported at laparotomy. Michel[6] reviewed 100 cases found in New Orleans. In Touro Infirmary, with 450 beds, there were 32 cases found in 3 years. The other 68 cases were discovered during a 10-year period at Charity Hospital, which has almost 3000 beds. Brooke[1] reviewed the cases of Meckel's diverticulum in Salt Lake City during a 10-year period. He reported 63 cases, or one in every 1000 laparotomies. In a recent study[5] of cases from the Children's Hospital in Pittsburgh, 63 cases were found in a 15-year period. These and many other reports emphasize the discrepancy between operative frequency and autopsy incidence.

It is evident, then, that for every diverticulum which is removed, there are 10 or 12 which are not discovered. Many of these are asymptomatic, but others undoubtedly produce symptoms which are not recognized. The obvious reason that more of them are not found at surgery is simply that an adequate search is not made. Certainly, there are times when a thorough examination of the small bowel is contraindicated but this is not often. A failure to search for the diverticulum when the appendix is normal or for other indications is inexcusable and at times is a fatal omission.

The racial incidence in this group is noteworthy because a diverticulum was not found in a negro patient. This curious fact has been noted in at least two other series. In the New Orleans Charity Hospital group[6] of 68 patients, to which reference has been made, a Meckel's diverticulum was found in negroes only 17 times, or 25 per cent. This is in spite of the fact that negro admissions have always exceeded white admissions by a large majority at Charity Hospital. In a recent series of 46 omphalomesenteric duct anomalies, Moore[7] was surprised to find no negro patients. The reason for the low incidence in the colored race is not apparent and it can only be concluded that it represents an example of divine discrimination.

The youngest patient was 10 years old and the oldest patient was 64 years of age. It is unusual that a diverticulum was not found in a child under the age of ten, for the condition is more often found in young children. Gross,[3] who reported 149 cases, found 45 per cent in the first two years of life. The only explanation for the higher age incidence in this group is the relatively small number of pediatric patients seen in the hospitals where this surgery was done.

A distinct surprise in the review of these cases was the sex distribution. There was only one male and fourteen females. This is at variance with the sex distribution in other series, large or small. Towbin[11] states that the incidence of Meckel's diverticulum in males is 3 or 4 times greater than in females, and Gross[3] found that 75 per cent of the diverticula occur in males. In the 100 cases reported by Michel,[6] 48 were found in males. The preponderance of females in this group is difficult to explain, but surely the distribution will change as more cases are added to this small series.

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CLINICAL FINDINGS

It should be recalled that these intestinal sacculations can vary tremendously in size and that the lining does not always consist of ileal mucosa alone. Gastric, duodenal, jejunal and colonic mucosae as well as pancreatic tissue can occur in various combinations within diverticula. Ten of these fifteen cases exhibited definite symptoms, and the other five cases were incidental findings at the time of pelvic surgery. This finding of 66 per cent for symptomatic cases is consistent with that found by others. Michel[6] found symptoms associated with 71 of the 100 cases in his series, and Kiesewetter[5] found a 60 per cent incidence of symptomatic diverticula.

The symptoms may become manifest in a variety of ways. Abdominal pain is the most frequent symptom associated with the lesion in the adult. The pain is caused either by inflammation of the diverticulum or by ulceration in aberrant gastric mucosa. The pain may be acute and severe, but usually is of the mild recurrent type. It may be generalized, but can become localized to the right lower quadrant. When this pain is accompanied by nausea, vomiting, fever and leucocytosis, it may be indistinguishable from appendicitis. Seven of the 15 cases in this series were operated upon with the diagnosis of appendicitis. One other patient had pain which simulated appendicitis, but the true diagnosis was suspected because appendectomy had been done four years previously. The important point to stress is that when the appendix appears essentially normal, further exploration is indicated. If a diverticulum is found both appendectomy and diverticulectomy are done.

Intestinal obstruction is not an uncommon complication of this anomaly, and it was this condition which led to the exploration of a 15-year-old patient in this series. A very large diverticulum had caused the adjacent ileum to be kinked by adhesions. There was impairment of blood supply and it was necessary to resect a portion of the ileum with the diverticulum. A Meckel's diverticulum can cause obstruction by other means, none of which were encountered in this group. One of the most common mechanisms is the invagination of the diverticulum to produce intussusception. A band extending from the diverticulum to the umbilicus, adjacent mesentery or another loop of bowel may cause obstruction. A rare cause of obstruction is the formation of an actual knot around the intestine by a very long diverticulum.

One of the most interesting cases in this group was a ten-year-old girl with an umbilical fistula. Past history revealed that she had had drainage from the umbilicus from birth until 6 months of age. The drainage then ceased until about three weeks before admission to the hospital. At that time she had a malodorous umbilical discharge with periumbilical cellulitis. After the infection had been controlled with hot compresses and antibiotics, excision of the umbilicus, the fistula and the diverticulum was done. A Meckel's diverticulum with an umbilical fistula is not a common variation of this anomaly. Gross[3] found only 11 cases in the 149 that he reported. The case just described offered no particular problem in diagnosis. However, if necessary, lipiodol can be introduced into the fistula to outline the tract. This should be done if one has reason to suspect a patent urachus.

There are other complications of a Meckel's diverticulum which were not found in this group. Perforation is not a rare complication. It may be caused by peptic ulceration or by penetration by a foreign body. Waldron[12] and Michel[6] each report a case in which perforation was caused by a fishbone. Malignancy in a Meckel's diverticulum is a rare occurrence, but both carcinoma and sarcoma have been reported. An unusual complication is Littre's hernia, in which a Meckel's diverticulum is the sole occupant of a hernial sac, either inguinal or femoral. Payson and associates[8] recently described a very unusual case in which a Meckel's diverticulum, strangulated in a femoral sac, showed a penetrating gastric ulcer.

The diagnosis was made preoperatively in only two of the fifteen cases. One of these was the child with the patent omphalomesenteric duct and the other was the patient who had had a previous appendectomy. The diverticulum had been overlooked at previous laparotomy in only two patients of this group. Occasionally a Meckel's diverticulum will be definitely suspected, looked for and not found. There are usually two reasons why these diverticula are overlooked. The inspection of the intestine has either been done hurriedly, or has not been continued far enough from the cecum. The lesion may be very small and can only be found by careful palpation with the fingers. Although diverticula usually arise from the anti-mesenteric border of the ileum,

they can occur elsewhere on the circumference of the bowel. The majority of the diverticula are found within the terminal three feet of the ileum, but they have been reported much farther from the ileocecal valve.

The type of tissue found in a diverticulum is of some interest, but it is not necessarily correlated with symptoms. It has been suggested that the more solid type of tissue, such as pancreatic tissue, might be more likely to initiate intussusception. No diverticulum in this group contained pancreatic tissue. Gastric mucosa was found in two of the diverticula and experimental studies have shown that this aberrant gastric mucosa is under the influence of the gastric antrum.

There is a difference of opinion concerning the advisability of removing a Meckel's diverticulum when it is an incidental finding. Some surgeons believe that such a diverticulum need not be removed, particularly if it has a broad base. This would appear to be unwise in view of the serious complications that can occur with this anomaly. Diverticulectomy can usually be done quickly and easily, and should be done unless it increases the operative risk. In the fifteen cases reported in this paper diverticulectomy was done in each case with no mortality and no complications.

Excision of a Meckel's diverticulum should be done in such a manner that the entire structure is removed without obstructing the intestine. Inversion with a pursestring suture is not recommended because it unduly constricts the lumen of the ileum. Most of the diverticula can be removed by applying two straight clamps obliquely at the base and cutting between the two. Closure is done with a continuous chromic suture, reinforced with interrupted fine cotton or silk. Occasionally a wedge excision will be necessary, especially if the base is thickened. Rarely, resection of the ileum with end to end anastomosis is the procedure of choice.

SUMMARY

A personal series of fifteen cases of Meckel's diverticulum is presented.

These fifteen cases were found in a four-year period, during which time 705 laparotomies were done. This incidence of 2.1 per cent is higher than that usually reported at operation. This frequency of discovery reflects an interest in and a search for this anomaly.

The group is unusual in some respects. The female to male ratio was 14 to 1. All patients were white and a Meckel's diverticulum was not found in a patient less than 10 years of age.

Seven of the 15 cases were operated upon with a preoperative diagnosis of appendicitis. Another had symptoms simulating appendicitis but had had an appendectomy four years previously. There was one patient with intestinal obstruction and one with a fistula at the umbilicus caused by a patent omphalomesenteric duct. The remaining five were incidental findings at the time of pelvic surgery.

Other complications, none of which occurred in this series are discussed, and the technique of diverticulectomy is outlined briefly. It is recommended that all diverticula be removed unless such a procedure would greatly increase the operative risk. This advice is warranted in view of the very serious complications which can occur with a Meckel's diverticulum.

1001 Main Street
Columbus, Miss.

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DOES TRANSFUSION CITRATE CAUSE HEMORRHAGE?

WILLIAM G. DIXON, M.D.

Provo, Utah

INTRODUCTION

The purpose of this experiment was to see if the wound oozing that is sometimes seen after multiple transfusions is caused by excessive citrate, as there is a tendency to blame bleeding on citrate poisoning. Various writers[5, 6, 7, 8, 11] have demonstrated that a hemorrhagic diathesis can occur from transfusions and that citrate toxicity can occur from transfusions[3, 4, 10] although no cause and effect relationship has been established between bleeding and citrate overdosage.

METHOD

The method of study was to rapidly inject intravenously large amounts of acid-citrate-dextrose solution (which is the anti-coagulant used in transfusions) into dogs and observe clotting times, calcium levels, tetany, pulse rates and blood pressures.

Acid-citrate-dextrose solution was used instead of blood because it eliminated the hemolytic reaction of blood itself. In addition the lesser amount decreased the danger of overloading the dog's blood volume, and it was more available than whole blood. The ACD solution, as prepared by Baxter Laboratories in Transvuso Vac blood donor bottles, contained 120 cc. of 1.37 per cent sodium citrate, 0.5 per cent citric acid, 2.45 per cent dextrose and water. This was considered equivalent to 2 gm. of disodium citrate in calculating doses. The ACD solution was rapidly dripped intravenously either into dogs anesthetized with Nembutal, 28 mg. per kg. of body weight, to simulate an operating room transfusion, or into conscious dogs to simulate the patient not under an anesthetic.

All clotting times were measured by the Lee-White method with saline rinsed 8-mm. tubes and later clotting times were also measured by the Lee-Vincent method with five drops of 1 per cent calcium chloride added to the tubes.[1] This latter method was used to measure lack of ionized calcium indirectly and qualitatively.

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An Emerson resuscitator connected to an endotracheal tube controlled respirations in the anesthetized dogs. A longitudinal sternal splitting incision afforded access for massaging the hearts and for recording the heart rates when peripheral pulses were no longer palpable. The thoracotomy incisions were a good test for oozing because no vessels were tied and only one or two were clamped. A mercury manometer connected to a 15-gauge needle inserted into the arch of the aorta measured blood pressures. The ACD was injected into the jugular vein of the anesthetized dogs and its rate of flow noted. Blood samples were taken from the inferior vena cava. Serum calcium was determined by the direct E.D.T.A. titration, using Calver 11 indicator powder obtained from the Hach Laboratories of Ames, Iowa.

In the conscious dogs the ACD solution was injected into one leg vein, blood samples were taken from another leg vein and the development and recovery from tetany was timed.

After the first few experiments it was found unnecessary to inject calcium chloride to restart the hearts and to relax the tetany because the excess citrate was quickly metabolized.

RESULTS

Results of the study showed that in 35 experiments injection of the ACD solution caused 26 cardiac arrests and 9 tetanies. The control coagulation times averaged 3 min. and 53 sec. in 20 determinations with the Lee-White method and 3 min. and 30 sec. in 11 determinations with the Lee-Vincent method. Clotting times before and after Nembutal anesthesia did not show any appreciable change in four dogs. During 31 injections of ACD solution Lee-White clotting times were prolonged 23 times, were the same two times, and were shortened six times.

Cardiac arrest occurred 26 times in 11 dogs, the heart being restarted with massage 15 times. Massage might have corrected more of the arrests except the arrest being used to terminate most of the experiments. Only one of the anes-

thetized dogs showed possible wound oozing, although the blood of almost all of the dogs failed to clot as the result of so much ACD solution. When blood pressure was measured in eight cardiac arrests, it fell 10 min. before the heart slowed. In all 26 cardiac arrests it was noted that the heart dilated and had a small weak beat for several minutes before the rate decreased (figs. 1 and 2).

Tetany occurred nine times in five conscious dogs injected with ACD solution. This was manifested first by sneezing and salivation and finally by laryngeal stridor and contraction of the legs. It was during tetany that the clotting time

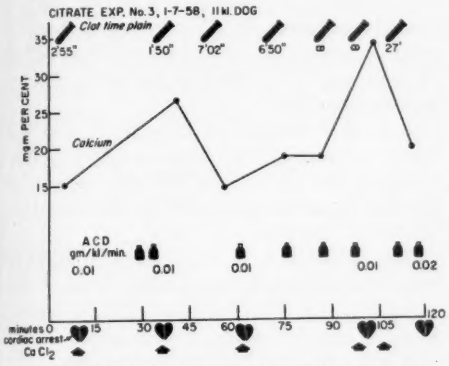

FIG. 1. ACD solution caused cardiac arrest five times and the heart was restarted four times with massage and calcium chloride. The clotting times and serum calcium levels rose intermittently.

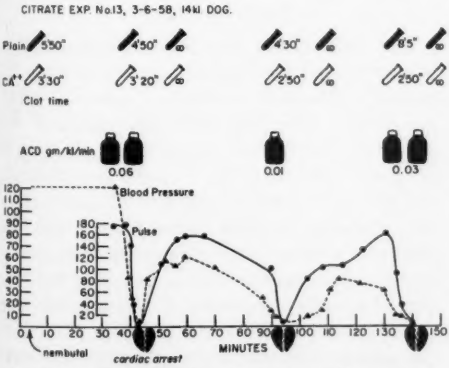

FIG. 2. ACD solution caused cardiac arrest three times and the heart was restarted twice with massage alone. The blood pressure fell before the pulse rate. The blood did not clot at the time of cardiac arrest but clotting soon returned to near normal on stopping the ACD.

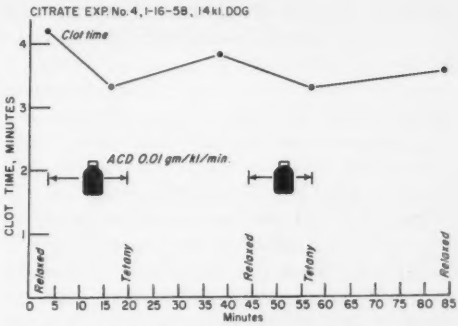

FIG. 3. ACD solution caused three tetanies which spontaneously disappeared on stopping the ACD. Clotting times showed a paradoxical shortening.

shortened, or showed the least prolongation. None of these dogs showed oozing from the vein puncture sites (fig. 3).

Serum total calcium levels rose an average of 3 mg. per cent after ACD solution had caused three cardiac arrests and one tetany. More determinations of calcium were prevented by hemolysis of the specimens which occurred with an unexplained high frequency.

In 28 measurements the dosage of sodium citrate to cause cardiac arrest or tetany in dogs averaged 200 mg. per kg. of body weight, or between 10 and 20 mg. per kg. per min.

DISCUSSION

Citrate toxicity. There is no doubt that large amounts of citrated blood have been given without causing tetany or cardiac arrest. Mollison[9] states, "Thus in an adult it appears safe to ignore citrate toxicity until a dose of 5000 ml. of citrated blood an hour is exceeded." The amount of citrated blood to be lethal, as determined by this experiment, would be about a pint a minute in an average man, but it is doubtful if this flow could be tolerated for very long. In spite of the large margin of safety of citrated blood, this margin is being exceeded with increasing frequency, as is shown by recent reports.[3] Cookson and associates[4] have cautioned against the danger of citrated blood in cardiac surgery especially under hypothermia. Nakasone and co-workers[10] attributed deaths from use of an artificial kidney to citrated blood and classified the electrocardiographic changes from it.

Calcium levels and functions. The total serum

calcium is about half diffusible calcium and half non-diffusible protein-bound calcium. Two-fifths of the diffusible calcium is ionized. Bruneau[2] noted the paradox of a rising total serum calcium in the presence of a citrate-induced tetany caused by the independent shifting of ionized and non-ionized calcium. The citrate mobilizes calcium from the skeleton, increasing the total serum calcium, but at the same time combines with the ionized fraction, reducing it to tetanic and asystolic levels. Citrate poisoning is not accompanied by bleeding because the amount of free calcium required for blood clotting is much less than the amount required to contract the heart and relax the skeletal muscles.[1] It is difficult to see how calcium injections can be of value in any bleeding problem, although Stefanini[13] observed that small amounts of calcium are necessary in almost all phases of the clotting process.

Another paradox of citrate and clotting noted in this experiment as well as by other investigators was the shortening of clotting times with smaller amounts of citrate. During the first hour of citrated transfusions Bruneau's[2] group found the clotting time shortened by almost three minutes. They stated this was first shown by Lewisohn in 1915 and was at one time advocated by Neuhof and Hirschfield as a therapeutic measure for internal hemorrhage. This surprising shortening of the clotting time has been attributed to injuries of the platelets[2] and to increasing the alkali reserve,[12] but these theories have not been widely accepted.

Transfusions and bleeding. Additional evidence that bleeding occurring from multiple transfusions is not caused by the citrate is in Howland's[6] report that the hemorrhagic diathesis occurred in spite of the fact that 1 gm. of calcium gluconate was given for every 1000 ml. of blood. Scott's group [11] studied the coagulation system of eleven battle casualties in Korea and found that generally clotting times were shortened, and that the platelet count and fibrinogen level were increased. No calcium lack was noted in performing prothrombin tests, and no hypocalcemic tetany was seen. There was slight oozing after transfusions of twenty pints or more, but no serious fault of hemostasis. McKay and co-workers[8] reported that the hemorrhagic tendency following an incompatible blood transfusion reaction was associated with thrombocytopenia, prolongation of coagulation time, decrease in circulating white blood cells and appearance of fibrinolytic activity and heparinoid anti-coagulant. Krevans[7] studied 32 transfused patients and found abnormal bleeding associated with thrombocytopenia in 11.

Heart dysfunction. In this study falling blood pressure was an earlier sign of impending cardiac arrest than the slowing of the heart rate. This fact is ignored by those monitoring devices for predicting cardiac arrest that only detect changes in heart rate and rhythm. Citrate hypotension has been reported by Strawitz and colleagues[14] in four battle casualties who received up to 12,000 ml. of blood and in whom the blood pressure did not rise until calcium gluconate was given.

SUMMARY

1. Transfusion citrate-ACD solution did not cause hemorrhage in 35 experiments, except possibly in one case, but did cause cardiac arrest 26 times in anesthetized dogs and tetany 9 times in conscious dogs.

2. In 28 determinations the amount of sodium citrate to cause cardiac arrest or tetany in dogs averaged 200 mg. per kg. of body weight, or between 10 and 20 mg. per kg. of body weight per minute of injection.

Acknowledgments. It is a pleasure to acknowledge the assistance of my wife, Harriet, the facilities of Dr. Tom Purvance's Alpine Animal Hospital and the chemical determinations by Mr. James Rawle of the Utah Valley Hospital.

35 North University Ave.
Provo, Utah

DISCUSSION BY C. C. SHULLENBERGER, M.D., HOUSTON, TEXAS

The study of the coagulation of blood in the laboratory and the application of the findings to clinical problems of bleeding is a matter of great complexity. Therefore, the employment of the most direct methods in making the application is of great value, and Dr. Dixon's investigation utilized such a direct approach. The anticoagulant property of sodium citrate depends upon its ability to combine with calcium and remove this all-important ion from its role as one of the king pins of the clotting process; if patients receiving massive doses of citrate were to bleed as a result one would, of course, expect that hypocalcemia and bleeding should coexist. Dr. Dixon's study, and others in a similar vein, indicate that hemorrhage is not a part of the clinical pattern of hypocalcemia. This is a valuable and necessary piece of information nowadays when a number of

surgical procedures are being introduced which require massive transfusion of preserved blood.

When unusual bleeding does occur under conditions requiring massive transfusion, there are some other causative factors to be thought of which bear some weight by virtue of experimental support. It is believed, for example, that anoxia (accompanying shock or difficulty with anesthesia) results in increased capillary permeability and generalized oozing hemorrhage. Under similar conditions, circulating anticoagulant substances of heparin-like quality have been demonstrated. Massive transfusions also are occasionally followed by a thrombocytopenic state, the mechanism of which is not understood, but which may be accompanied by hemorrhage.

I believe continued investigation will show that these, plus perhaps other factors not yet elucidated, will account for most of the bleeding problems which have been attributed to transfusion citrate.

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REHABILITATION OF THE PATIENT WITH HEMIPLEGIA*

DONALD A COVALT, M.D.

New York, New York

Hemorrhage, thrombosis, embolism, trauma, and congenital anomalies such as ruptured aneurysms of the cerebral vessels are the causes of cerebrovascular accidents. The above are listed according to their incidence so that hemorrhage causes the greatest number. However, the greatest number of patients who seek rehabilitation will be suffering from thrombosis of the cerebral vessels because approximately fifty per cent of patients who have a cerebrovascular accident caused by hemorrhage do not survive.

The saving of his patient's life is the major concern of the physician-in-charge. If he accomplishes this, then he must begin early to think of how to prevent the contractures and atrophy of muscles that develop so rapidly in these patients.

One of the most important aspects of this is the proper positioning of the paralyzed extremities; this can save weeks, perhaps even months of physical therapy. Utilization of small pillows will keep the afflicted arm away from the chest. The use of a greater number of small pediatric pillows rather than a few large adult pillows is much better. The arm should be positioned so as to prevent contracture of the shoulder. Holding of the arm close to the chest with inward rotation always occurs in these patients. Use enough pillows to keep the arm well away from the chest so that each joint will be positioned higher than the preceding one. If this is done correctly, the elbow joint will be higher than the shoulder joint, the wrist joint will be higher than the elbow, and the fingers will be higher than the wrist joint. A small roll of bandage with padding should be used to keep the thumb away from the hand with the fingers slightly flexed. This positioning of the upper extremity will help to prevent edema of the hand seen so often in these patients.

Professor, Department of Physical Medicine and Rehabilitation, New York University College of Medicine; Associate Director, Institute of Physical Medicine and Rehabilitation, New York University-Bellevue Medical Center, New York, New York.

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It is also important that the lower extremity be positioned. Without this, the leg will fall into outward rotation with a toe drop. A neatly made and tightly boxed bed, while tidy looking, should not be used. Sheets should be loose and should not drag on the toes. Sand bags are placed at the thigh and below the knees on the lateral aspect of the leg which will keep it in a neutral position. Additional sand bags can be used to maintain the foot at a right angle to the leg; this will prevent toe drop caused by shortening of the gastrocnemius muscle. A foot board may be used to maintain the foot at a ninety degree angle. However, the sand bag is usually easier to obtain and is probably more satisfactory. When the above positioning has been accomplished, the patient is then in an ideal position to prevent complications. Minor adjustments can be made occasionally to make the patient comfortable.

It is up to the clinician to decide when the patient's condition will tolerate a more active program. It has been our experience that patients with a hemiplegia caused by hemorrhage usually can be started on a more active program within a week after the initial onset; patients with an embolism or a thrombus usually can be begun earlier—within two or three days. Each case must be individualized, of course, and only the patient's physician can make the final decision for a more active program.

Registered physical therapists, if available, should be utilized because of the experience and training that they have had in treating this type of case. However, a nurse or member of the family can be taught to carry out the rather simple exercises that are essential in maintaining the tone of the muscles, preventing contractures, and increasing the strength of weakened muscles.

A very detailed prescription of the exercises and stretching that is desired must be written out by the physician-in-charge.

The first step by the therapist should be the taking of the paralyzed extremities through a complete range of motion of each joint starting with the upper extremity. In other words, the

shoulder is taken through a complete pattern of range of motion, including flexion, abduction, and adduction, internal and external rotation. When this has been completed, the shoulder should then be taken through a complete circumduction. The arm is brought to the side and the elbow is flexed and extended. The lower part of the arm is then placed in supination and taken through to pronation and return. The hand is flexed at the wrist joint, extended, and brought back to a neutral position. It is then moved medially and again laterally. The finger joints must be taken through all ranges of motion. The therapist does all these exercises—passive motion.

Next comes the lower extremity, starting usually with the hip joint. The leg is taken from adduction to abduction (with the leg flexed at the hip joint) and brought back to extension. The leg should be taken through internal and external outward rotation; the leg is then raised and the knee joint is flexed and brought back to extension. The ankle joint is taken through all of its motion with particular attention paid to the dorsi-flexion of the foot. Stretching of the heel cord is accomplished by cupping the patient's heel in the therapist's hand and placing her forearm against the ball of the foot exerting enough pressure to stretch the heel cord and to bring the foot up to at least a ninety degree angle and if possible, beyond that. It is recommended that these exercises should be carried out twice a day —once in the morning and once in the afternoon.

The program should be increased in a few days as the patient improves, and he should be encouraged to help in these exercises; for example, in using the elbow, the patient should be requested to attempt to flex the elbow as far as possible. Ask the patient to bring his hand to his mouth. The therapist should hold the arm in such a way as to do away with gravity so as to help the patient in flexing the elbow. If he finds it impossible to complete the entire range of motion, the therapist will then complete the motion. Then the therapist should ask the patient to attempt to stretch the elbow out in the same manner as stated above, and if he fails to complete this motion, the therapist must go through the entire range of motion again. Every joint on the affected upper and lower extremity should be put through this exercise.

The patient must be encouraged to use more and more of his own muscle power as his muscle power returns and he becomes more alert.

Once again, it is up to the physician to decide how soon his patient can be allowed to sit up. Usually in the older aged group, these patients are apt to lose their sense of sitting and standing balance very quickly unless they are brought to a sitting position as soon as possible. When receiving permission from the physician, the therapist will help the patient to sit up, first with his feet in bed, and later, by dangling his legs over the side of the bed. An important factor is to have the patient in a low bed instead of a high bed. For the patient who has had a stroke, there is nothing worse than to find himself sitting on the edge of the bed with his feet dangling over a little box and finding the floor to appear to be miles away. The patient should be put in a low bed with his feet firmly on the ground and spaced widely apart. In teaching him sitting balance, his good arm should be extended, his hand flat on the bed, and his two feet on the ground with the therapist standing by his affected side. He can be allowed to quickly increase his sitting time from five to ten minutes. These exercises and the sitting position should be performed twice a day, once in the morning, and once in the afternoon.

Again, it must be emphasized that it is much better for the patient to sit on the edge of the bed preserving his balance with his unaffected arm than it is to seat him in a more comfortable armchair where he does not have to do any work. The therapist must take care to see that the patient does not fall. Ordinarily, the sitting time can be increased rapidly.

The physician must give the orders for the patient to be allowed to try to stand. Ordinary ladder back kitchen chairs are very convenient for this purpose. They are placed at the bedside with their backs towards each other and the patient sitting on the edge of the bed between them. If complete paralysis of the hand is present, it will be necessary to bind it lightly to the top of the back of the chair. The therapist should take her position in front of the patient making sure that the patient's feet are spaced widely apart. The therapist exerts pressure against the patient's knee to prevent it from buckling. With the therapist continuing to stand in front of the patient, he is encouraged to maintain standing balance. The standing period may be of a very

short duration to begin with, but it can be increased rapidly until it is felt that no danger can be incurred if the patient stands by himself; however, this must always be while the therapist is present.

A manual muscle test of the upper and lower extremities should be done at this time to determine the type of bracing necessary. For example, if the quadriceps muscle is weak or absent, a long leg brace will be necessary. A double bar long leg brace with box lock at the knee joint on the medial bar with a stirrup attachment and a ninety degree stop at the ankle joint should be the prescription. The majority of these patients have weakened and absent musculature below the knee usually resulting in a toe drop and an inversion of the foot, even if the quadriceps muscle is strong. A short leg brace should be prescribed as follows for these patients: Double bar short leg brace with a stirrup attachment at the ankle joint and a ninety degree stop.

Upon delivery of the brace, the time has come for the patient to learn to walk and again the ladder back kitchen chairs can be utilized. The patient should be instructed to walk in a reciprocal manner. The therapist takes her position beside the patient on his affected side. For example, if we have a patient with a right-sided hemiplegia, the therapist stands beside the right chair and moves the chair ahead approximately one foot and requests the patient to bring his left leg through. It should then be possible for the patient to move the left chair forward with his unaffected left hand, meanwhile taking a step at the same time with his affected right leg. This is the normal manner of walking. As the left leg swings through, the right arm swings forward. Parallel bars can be utilized if they are available.

The patient should be able to be transferred from the chairs or the parallel bars to a cane and to walk with assistance on a flat level surface within a matter of a few days or a few weeks. The patient also must be instructed on how to climb curbs and steps.

Muscle re-education for all muscles in the afflicted extremities of fair grade or less and resistive exercises for all muscles of fair plus and above should be included in the doctor's prescription.

For those patients who do not completely recover and a majority do not, the "activities of daily living" must be taught. Many patients having a completely paralyzed upper extremity have been taught to feed themselves, dress themselves, take care of their personal hygiene, ambulate and become completely self-sufficient. The amount of residual paralysis is the factor that determines the amount of instruction necessary.

In those patients with speech involvement, this can best be taken care of by a competent speech therapist. If one is unavailable, a book published by the Institute of Physical Medicine and Rehabilitation, of the New York University-Bellevue Medical Center, entitled "Aphasia Rehabilitation: Manual and Work Book" can be obtained and utilized by a nurse or even a member of the family.

Tremendous emotional problems are present in many of these patients. Such a disaster is often followed by a natural depression, but they will respond to understanding and a lot of "tender loving care." The patient himself is the most important member of the rehabilitation team and he and his family must be made to understand this. What the patient learns to do for himself and not what the doctor, the therapist or the family do for him is the most important part of his therapeutic program.

Many patients have a tendency to laugh or cry at the slightest provocation during the early stages of the illness. This is perfectly natural and it should be stressed to the patient and his family that this manifestation will improve as the patient improves. It is a thalmic disturbance which usually disappears entirely.

The majority of patients with hemiplegia should be fully trained within a couple of months. Of course, more severely paralyzed patients take longer.

SUMMARY

Careful positioning of the paralyzed extremities should be started early. Within two to six days of onset, passive, then active assistive exercises can usually be started. In order that the patient can be taught to walk as soon as possible, sitting and standing balance should be started early. The patient must be instructed to care for his own "activities of daily living." Many patients with hemiplegia will need speech therapy.

Lastly, the fact that the patient must be the most active member of the rehabilitation team is of great importance.

Institute of Physical Medicine and Rehabilitation
N. Y. University-Bellevue Medical Center
New York, N. Y.

INTRACRANIAL EPENDYMOMAS AND THEIR SURGICAL TREATMENT

E. LYLE GAGE, M.D.

Bluefield, West Virginia

The intracranial ependymomas represent approximately 5 per cent of intracranial gliomas.[12, 5, 7, 8] They are derived from the ependyma or the lining cells of the fluid containing cavities of the brain and spinal cord. Their surgical treatment has been discouraging because of their sites of origin, their size and their attachment to, or infiltration into, vital structures of the nervous system.[11, 5, 3]

In an attempt at more thorough understanding of the growth and behavior of these tumors, and in the hope that by knowing more about them some might prove removable or curable, a study has been made of our patients and a review of seven cases is presented.

In embryonal development the ependyma are the earliest cells to differentiate from the neural tube.[11, 9] They arise from the primordial spongioblasts which remained close to the internal limiting membrane.[9, 11] The ependymal cell is the only cell of the central nervous system which retains its epithelial characteristics, and it is closely related embryologically to the germinal cells of the multipotential syncytium-like, mantle layer.[11]

This is the type cell of the tumors known as ependymomas which are considered in this paper.

This report is based upon seven patients with ependymomas whom we have examined at the Bluefield Sanitarium. Six were operated upon and one died in a convulsive seizure before studies were complete or operation could be performed. Each patient represents a different type, location or behavior of the growth and each illustrates certain points of interest concerning the treatment, operability, and prognosis.

Case 1. E. K., a 27-year-old colored waitress, had headaches for six months before admission. On examination she showed an unsteady gait, weakness of the left hand, ataxia, a right Babinski sign and left hypesthesia below the face. She had lateral nystagmus and some loss of vision. A ventriculogram showed enlarged lateral and third ventricles.

At operation, a small oval intramedullary tumor six millimeters in diameter was found in the center of the medulla oblongata, 9 mm. caudad from the calamus scriptorius. The tumor bulged dorsally under the arch of the foramen magnum causing some obstruction. An incision in the midline of the medulla allowed the tumor to extrude into the operative field and it was attached to the central canal by a pedicle. When traction was made on the pedicle the heart rate dropped from 80 to 47 beats per minute. Release caused return of the heart rate to normal. The traction was repeated with the same result. The idea of dissecting out the attachment of the pedicle was given up, and the tumor was removed cauterizing the pedicle down to its attachment in the central canal. The tumor was a moderately uniform cellular ependymoma with moderate stroma and with no cysts, calcium, mitoses or vascular abnormalities (fig. 1).

The patient resumed her work as a waitress for four years and then reported by letter that she was becoming paralyzed five years after operation.

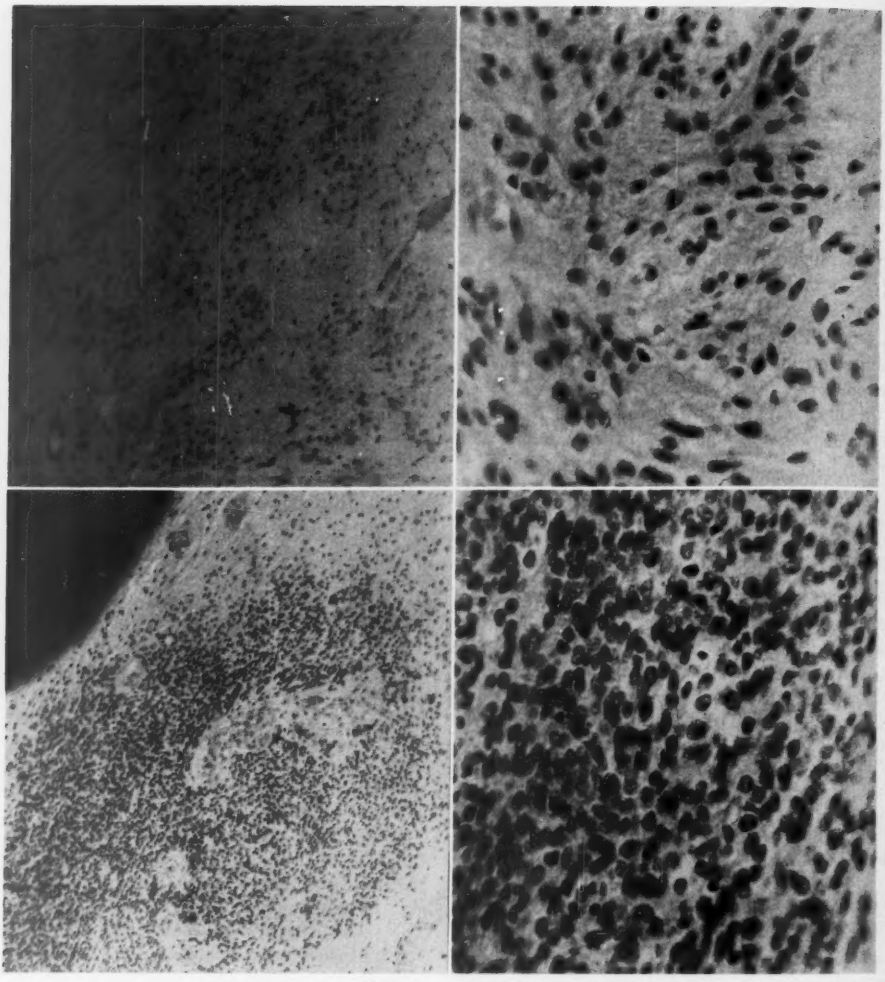
Case 2. Mr. C. M., age 49, complained of productive morning cough, sinus trouble, unsteadiness for one year, and headaches for three years. He had an unsteady gait, lateral nystagmus to both sides, early papilledema, an opaque right antrum and a productive rattling cough. A ventriculogram showed dilated ventricles, but no visible aqueduct of Sylvius. X-ray of the chest revealed no pulmonary lesion.

At operation a large midline posterior fossa tumor, reddish grey in color was found separating the tonsils of the cerebellum and extending cephalad to the opening of the aqueduct of Sylvius. The entire tumor was removed and the pedicle, which consisted of a single strand of tissue with a large artery, was cut off flush with the floor of the fourth ventricle. The artery apparently had supplied the tumor.

This ependymoma was moderately cellular with cysts scattered through it. There was abundant stroma but, no pleomorphism, and no mitotic figures were seen (fig. 2).

The patient did well and his hoarseness, cough, and unsteadiness cleared.

Case 3. Master W. R. W., age 6, had a cold and what was diagnosed elsewhere as meningitis, with 887 white cells in the spinal fluid. Three months later he was admitted to our service with all of the signs of a midline cerebellar tumor and blindness. At the insistence of the boy's parents, a suboccipital craniotomy was performed. A large bulging greyish red tumor filled the entire fourth



FIGS 1, 2, 3 and 4
FIG. 1 (*top left*); FIG. 2 (*top right*); FIG. 3 (*bottom left*); and FIG. 4 (*bottom right*).

ventricle displacing laterally both cerebellar lobes and taking the place of the vermis. The tumor was attached over a wide area to the entire floor of the fourth ventricle. Several grams of tumor were removed until spinal fluid finally came forth from the aqueduct into the posterior fossa.

The boy recovered from the operation, but remained blind and somewhat ataxic until his discharge after 4000 r of x-ray therapy.

Five and one-half months later, he returned with evidence of regrowth of the tumor. A second operation was performed and an attempt was made to peel the tumor from the medulla. This was impossible and excessive bleeding, respiratory difficulty and fall in blood pressure resulted. The bleeding was controlled and vital signs were stabilized, but two days after operation he developed uncontrolled hyperthermia and died.

This tumor was a broad-based, cellular, mixed type neoplasm and in one place showed a nest of more malignant closely packed cells (fig. 3) with several mitotic figures (fig. 4). In other areas there were small bits of calcium as well as multipotentiality and variability of cell behavior. There was some hyperplasia of the vascular epithelium almost like that seen in glioblastomas, yet the tumor

was grossly and microscopically classified as an ependymoma.

Case 4. J. R. M., a five-year-old white boy began to complain of headaches three weeks before admission, and he had vomited several times. He had a large head and two diopters of papilledema, but the examination otherwise was normal. An x-ray of the skull showed some separation of the suture lines, a lumbar puncture revealed an initial pressure of 150 mg. of protein. A ventriculogram revealed evidence of a large tumor in the right frontal region with obliteration of the anterior portion of the right lateral ventricle. At operation, a large nodular tumor presented itself 2 cm. beneath the cortex of the right frontal lobe. Several grams of the tumor were removed and it was believed that the foramen of Monroe was decompressed. Because of variable vital signs the operation was terminated. A second operation was performed and the remaining tumor removed as completely as possible, including a large piece in the third ventricle. The only remaining visible tumor was an infiltration about arteries at the base of the brain.

This growth was a very cellular ependymoma with pseudorosettes and a cyst, but mitoses were not definitely found. The child was treated with 3,500 r of x-ray.

The boy returned home (fig. 5) and resumed his schooling very efficiently until over three years later, when he returned with a sudden onset of right costal pain, nausea and vomiting. A myelogram showed a diffuse lesion from the 9th dorsal to the lumbar cord.

The autopsy showed an extensive intra-ventricular ependymoma with seeding along the spinal cord.

Case 5. W. E. C., a 52-year-old railroad electrician, for five or six months had noted headaches associated with dizziness and falling spells described as convulsions. He had not lost consciousness, but he described a trembling for three or four minutes associated with each spell. Examination revealed an old left peripheral facial palsy, 2 diopters papilledema, poor co-ordination, and a left Babinski sign. X-ray of the skull showed calcification of the pineal gland displaced forward and upward. A ventriculogram showed markedly dilated lateral ventricles and no definite third ventricle, yet we decided that a posterior fossa tumor was present.

FIG. 5

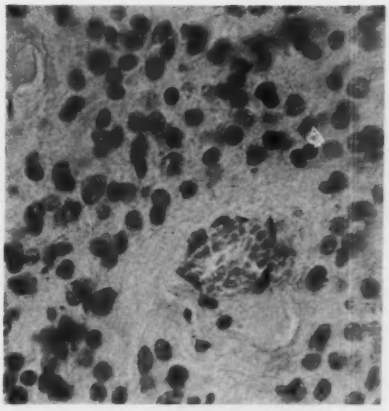


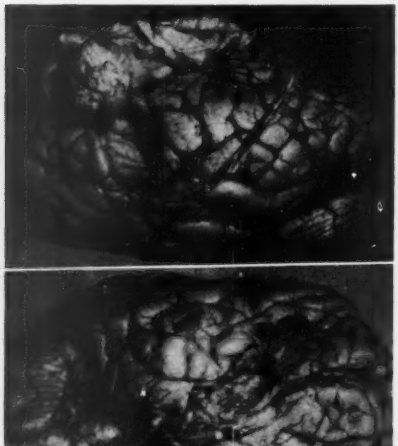
FIG. 6

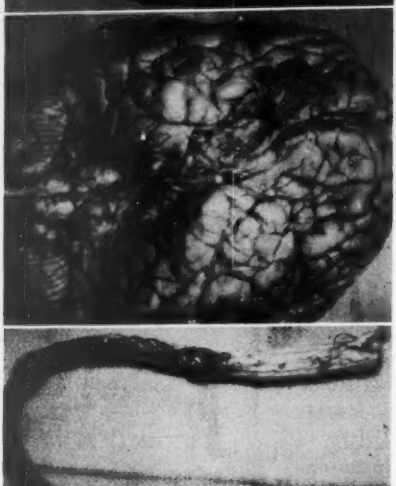
With a suboccipital craniotomy, a red beefy tumor coming up through the left cerebellar lobe was exposed. It was quite hard and nodular and was removed completely in three large pieces. Brisk bleeding was encountered as the last piece came away. This bleeding was from an artery along the brain stem which apparently had supplied the tumor. This artery was cauterized and no remaining tumor could be seen.

This ependymoma was of the epithelial type consisting of nests of cells with some central canals, a delicate stroma, and numerous capillaries (fig. 6). The cells showed indistinct cytoplasm and regular homogenous blue nuclei, which were occasionally hyperchromatic. There were no mitoses or pleomorphism.

The patient recovered and was symptom free in December 1957.

Case 6. Mrs. J. W. H., a 54-year-old widow had complained of migraine headaches, and contact dermatitis on previous visits to her doctor. She had suddenly developed pain and sensory disturbance in her left leg and hip. Two days later she was admitted to our service, where she was found to show drowsiness, no papilledema, normal





FIGS. 7, 8 and 9
FIG. 7 (*top*); FIG. 8 (*center*); and FIG. 9 (*bottom*).

cranial nerve signs as far as could be tested, normal symmetrical deep reflexes, and an equivocal plantar response on the left side, with a right Babinski sign. There was a bizarre and irregular loss of sensation to touch, pain, and temperature over the legs and trunk. Medical consultation revealed no explanation for the signs and symptoms.

A lumbar puncture revealed 130 mm. of water pressure, and very gentle jugular compression caused a rise to 300 mm. of water with prompt fall to normal. The spinal fluid revealed 150 mg. per cent of protein, 4 plus globulin, 4 polymorphonuclear cells, 1 lymphocyte, and negative serology.

A myelogram was planned for the next day but, the patient had a sudden convulsion and died abruptly after eating her supper. The autopsy revealed a left parietal meningioma, and a right basilar malignant ependymoma with beadlike seeding along the entire spinal cord (figs. 7, 8, 9).

The presence of tumors from different embryonic cell layers in different hemispheres at the same time is unusual.

Case 7. Mr. J. B., age 20, had been troubled with pain in the chest and weakness for five weeks. His feet and legs had been numb and weak for two weeks and he had been paralyzed in the lower extremities for five days. Examination revealed a congenital divergent squint, normal cranial nerves otherwise, no papilledema, no cerebellar signs, paralysis of the lower extremities, and a level of anesthesia to thoracic 6 on the left, and thoracic 7 on the right. He had a complete block in the spinal canal and a myelogram indicated a tumor at the level of thoracic 6.

On December 5, 1956 an elongated tumor involving the spinal cord at thoracic 6 was removed and it was an anaplastic, vascular ependymoma, with numerous mitotic figures. Following its removal he was treated with 3500 r of x-ray.

The patient learned to walk with a cane and came back to show us his progress. Five months later he returned on a stretcher with headaches, loss of memory of one week, 3 diopters papilledema and signs of a posterior fossa tumor. At operation, we found a large beefy tumor growing up through the left lobe of the cerebellum. Great quantities of the tumor were removed until the base attachment was found to extend over a long area of the brain stem in the so-called gutter area. The tumor was similar to the one in the cord. The boy recovered and resumed ambulation.

Four months later he returned and a second suboccipital removal of large quantities of tumor was carried out. Symptoms subsided, the patient went home and resumed a life of comfortable wheel-chair living; also he could walk with help.

Three months later the patient returned in critical condition, with nodular regrowth palpable beneath the suboccipital scalp. He could not eat or retain liquids. At this stage he was given a course of 4,000 r of x-ray therapy to the posterior fossa. The tumor melted away as though by magic and the boy became his smiling jovial self again. He was able to walk with a cane. After another three months the symptoms returned and he died.

Dr. Harvey Cushing reported a 31.8 per cent mortality with patients having intracranial ependymomas. Bailey, Buchanan, and Bucy[3] wrote, "The ependymomas are frankly disappointing. We have a 50 per cent mortality." Troland, Kendricks, Sahyoun, and Manderville[11] grouped ependymomas into grades 1, 2, 3, and 4,

as regarding their potentiality and behavior. After reviewing these seven cases it would seem that there is a direct correlation between the degree of malignancy, the infiltration, size of the area of attachment, and the inoperability of the ependymomas of the posterior fossa. The one right frontal tumor was cellular and showed infiltration about blood vessels with seeding to the cord, yet palliative operations permitted three and a half years of comfortable happy life for the patient.

If any conclusion can be drawn from but seven cases it must be that the operability is inversely proportional to the breadth of attachment, the multipotentiality of the cells and the evidence of malignancy.

X-ray therapy was certainly effective in reducing the size of the recurrent tumor in case 7 and it may have retarded regrowth in some of the other patients.

In removing these tumors great care should be exercised not to leave any particles of tumor attached to the tissues of the nervous system as they will remain there for seed.

It is believed that the ependymomas of less cellularity and small attachment *may* be completely removed with care and caution. Any attempt at *complete* removal of those tumors with a broad attachment appears unjustified, but most of the patients can be relieved of their acute symptoms by judicious surgery and x-ray therapy.

Bluefield Sanitarium Clinic
Bluefield, West Virginia

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CHANGING PATTERNS OF SURGICAL PRACTICE

CARROLL H. LONG, M.D., AND GILBERT A. RANNICK, M.D.

Johnson City, Tennessee

Two related medical needs have been recognized in recent years. The first involves chiefly the medical schools and emphasizes the necessity for training a greater number of physicians for an enlarging population and for finding more clinical material for teaching purposes in a prosperous economy. Prosperity has been something more than a blessing to medical education, for it has so lessened the number of "service" patients that residency programs have suffered. Complete medical indigency has become unusual and will become even more so if plans of labor unions and well meaning bureaucrats are carried through. Recently theories have been advanced placing upon certain private patients the duty of submitting to surgery by the trainee member of the surgical team as a matter of the individual patient's "responsibility" to medical science. At one medical center surgeons are urged to persuade a portion of their private patients to accept the resident as the operating surgeon. In another teaching hospital a patient with no medical resources other than a single insurance policy providing hospital and surgical benefits is classified as a service patient. But these and other stratagems do not replace the myriad service patients found in the 20- to 40-bed wards of another day.

The second medical need upon which attention has been focused recently is for the provision of the best medical care for all citizens whether or not they live in medical centers. This matter has become acute since equality of medical service now seems an attainable goal largely because of the recent subsidizing of hospital construction and the increasing number of well-trained specialists who are finding permanent locations in widely dispersed regional hospitals.

It will become more obvious that there is a relationship between these two medical necessities if it can be shown that the medical schools' need for more clinical material and expanded training facilities can be met in part in the regional hospitals and if it can be shown that the quality of medical practice in the hinterland can be raised by medical school faculty collaboration and supervision of training programs in the regional hospitals. Several studies are in progress which are producing pertinent information.

The Governors Committee of the American College of Surgeons[3] is engaged in a study of the elevation of standards of smaller hospitals and the care of the surgical patient in smaller hospitals. The study has progressed far enough to indicate that "a striking amount of work is done in these smaller hospitals under 50 beds" and that "there are definite areas in which the American College of Surgeons can assist in the elevation of standards in the smaller hospital." Under the direction of Dr. Frederick A. Coller[1] a study is being made in Michigan, Oklahoma and Massachusetts using Blue Shield statistics to show the training of surgeons performing various types of surgical procedures. The Bureau of Medical Economic Research of the American Medical Association[2] has made an ambitious study of some categories of patients treated by physicians in hospitals which presents a multitude of data in this field.

Several well planned collaborating efforts have been made by medical school faculties and community or regional hospitals to solve their mutual problems. At the Hunterdon Medical Center in Flemington, New Jersey, the New York University Medical School[4] maintains such a relationship with the rural hospital staff that each of the full time specialist directors of service at Flemington is a faculty member of the Medical School. It is postulated that this relationship continues the medical training of the general practitioner members of the hospital staff and supplies the rural community with medical care by medical school faculty specialists.

"The New York University Plan" of the New York University Post-graduate Medical School[6] is "an example showing the potentialities of regional hospital-medical school affiliation." At present it is limited to six hospitals (within a radius of 50 miles of the school) which are approved for intern or resident training. The medi-

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SURGICAL PRACTICE SURVEY QUESTIONNAIRE

		1946			1956		
I.	TOTAL HOSPITAL BEDS						
II.	TOTAL NO. PROFESSIONAL STAFF (M.D.)						
III.	TOTAL OPERATIVE PROCEDURES		MAJOR	MINOR		MAJOR	MINOR
IV.	BOARD CERTIFIED SURGICAL SPECIALISTS	OPERATIONS PERFORMED			OPERATIONS PERFORMED		
		NO.	MAJOR	MINOR	NO.	MAJOR	MINOR
	(a) General Surgery						
	(b) Orthopedic Surgery						
	(c) Urological Surgery						
	(d) Thoracic Surgery						
	(e) Neuro Surgery						
	(f) Ophthalmology						
	(g) Otolaryngology						
	(h) Gynecology						
V.	BOARD ELIGIBLE SURGICAL SPECIALISTS						
	(a) General Surgery						
	(b) Orthopedic Surgery						
	(c) Urological Surgery						
	(d) Thoracic Surgery						
	(e) Neuro Surgery						
	(f) Ophthalmology						
	(g) Otolaryngology						
	(h) Gynecology						
VI.	GENERAL PRACTITIONER SURGEONS						
VII.	SURGICAL RESIDENTS						
VIII.	INTERNS						
IX.	MEDICAL STUDENT EXTERNS						

FIG. 1

cal school participates in the training program by frequent faculty visits to each hospital and encourages attendance of the regional hospital staff members at seminars, clinics and conferences at the medical school.

The Faculty of the University of Michigan Medical School[5] is engaged in a major activity in the field of postgraduate medical education in certain regional hospitals where it makes the physician a teacher in his own institution. Under close supervision by the parent faculty, 17 hospitals over the state are carrying out parts of the training of University of Michigan residents in surgery, medicine, gynecology and obstetrics and in general practice. Two hospitals are carrying out part of the training of undergraduate medical students. The program is said to be highly successful and fulfills several objectives: the best in postgraduate training for the regional hospital staff member through his intimate contact with trainees and faculty advisors, the provision of varied and plentiful clinical experience for the resident and student and, lastly, the completion of the regional hospital surgical teams with competent house officers. Particular attention is called to this provocative report.

The study here reported was undertaken in an effort to develop information which would in-

crease the understanding and hasten a solution of the problem already outlined. Data were sought concerning surgical practice experience in Tennessee hospitals during 1956. Questionnaires were sent to all general hospitals and personal interviews were held in many instances (fig. 1). Useful data came from two groups: small community hospitals, state wide in distribution, with bed capacities of less than 80 beds, and a small group of "regional hospitals" located in upper East Tennessee, which serve a population of 500,000.

THE SMALL GENERAL HOSPITAL

There are 135 small general hospitals in Tennessee with bed capacities of less than 80 patients each, having a listed total bed capacity of 3,766 (table 1). Useful data were received from 63 hospitals in this group (47 per cent) which had a combined bed capacity of 1806 patients or 49 per cent of all the beds in this group of Tennessee hospitals.

Surgical procedures were performed by 279 surgeons in these 63 hospitals during 1956 (table 2); 244 (87 per cent) were general practitioner surgeons and 35 (13 per cent) were Surgery Board Diplomates or Board Qualified surgeons. The 35 well-trained men were distributed in 17 hospitals (27 per cent).

In the responding 63 small hospitals during the year 1956, 10,470 major operations were performed, of which 1,903 (18.5 per cent) were done by Surgery Board Diplomates or Board Qualified surgeons and 8,567 (81.5 per cent) by general practitioner surgeons (table 3). During the same period, 15,536 minor procedures were performed, 4013 (26 per cent) by Board Diplomates or Board Qualified surgeons, the remainder by general practitioner surgeons.

It is interesting to note that 46 per cent of the 63 responding small hospitals are not accredited by the Joint Commission on Accreditation of Hospitals and that 2768 major surgical procedures, or 26 per cent of the total number of major operations performed in this group of small hospitals, were done in non-accredited institutions (table 4).

These data are thought to be representative of the small hospitals in Tennessee and indicate the large volume of surgery performed in these institutions by general practitioner surgeons, corresponding in general with observations made by

TABLE 1

Small hospitals—Tennessee (1956)

	No.	*Per cent*
Hospitals polled	135	
Responding	63	47
Bed capacity		
All hospitals	3766	
Responding	1806	49

TABLE 2

Small hospitals—Tennessee (1956)

	No.	*Per cent*
No. hospitals	63	
No. surgeons (total)	279	
Hospitals with Board or Board Qualified surgeons	17	27
Board or Board Qualified general surgeons	35	13
General practitioner surgeons	244	87

TABLE 3

Operations performed in small hospitals in Tennessee in 1956

	No.	*Per cent*
Total number of major operations performed	10,470	
Major operations performed by Board or Board Qualified surgeons	1,903	18.5
Major operations performed by general practitioner surgeons	8,567	81.5
Total number of minor operations performed	15,536	
Minor operations performed by board or Board Qualified surgeons	4,013	26
Minor operations performed by general practitioner surgeons	11,523	74

TABLE 4

Small hospitals—Tennessee (1956)

	Number	*Per cent*
Hospitals accredited by J.C.A.H.		54
Hospitals not accredited by J.C.A.H.		46
Major operations performed in accredited hospitals	7702	74
Major operations performed in non-accredited hospitals	2768	26

the Governors Committee of the American College of Surgeons[3] in Nebraska, New Jersey, Texas, Missouri and Montana. The number of Board Diplomates and Board qualified surgeons found on the medical staffs of small Tennessee hospitals is encouraging but not of controlling importance at this time. Perhaps the most provoking observation is the number of these institutions which are not accredited and are therefore without extrinsic professional control. It is suggested that small hospital accreditation be on the basis of a sliding scale, dependent in part upon local law and customs and in part upon the practical possibilities of a small organization. Encouragement of the small legally licensed non-accerdited hospital to seek accreditation on terms it can accept will increase extrinsic professional control in these hospitals, now non-accredited, where so large a segment of the population seeks surgical coverage.

The public is becoming alert to its right to better medical care and more vigorous leadership in this direction should be exerted by local and state medical organizations. The tendency for physician-owners of small hospitals to resist replacement by larger non-profit community hospitals is encouraged by the present policy of withholding state and federal aid in hospital construction from communities in which the aggregate number of hospital beds seems sufficient. The small community inevitably benefits from the organization of a single larger non-profit hospital which provides extrinsic professional supervision through accreditation and protects its patients by justifying the privileges of its medical staff members by fixed criteria.

THE REGIONAL HOSPITAL

We defined a regional hospital as one which—because of its size and the excellence of its organization and medical services—attracts patients from a region rather than from a local community. In an effort to find accuracy in data from a representative group of regional hospitals we have chosen those located in upper East Tennessee, an area which with its surrounding market territory of western North Carolina and southwest Virginia has a population of approximately 500,000 people. In it are three relatively large general hospitals and a Veterans Administration Hospital with a combined bed capacity of 1175. Our data from these institutions are complete (table 5).

TABLE 5

*Capacity of regional hospitals in upper East Tennessee in 1956**

Hospital	No. of Beds
Johnson City (Memorial)	187
Kingsport (Holston Valley)	245
Bristol (Memorial)	143
Mountain Home (Veterans Administration)	600
Total no. of beds	1175

* 100% response.

TABLE 6

Surgeons in regional hospitals in upper East Tennessee in 1956

	No.	*Per cent*
Board or Board Qualified surgeons	51	64
General Surgeons	23	29
Sub-specialists	28	35
General practitioner surgeons	29	36
Total	80	

TABLE 7

Major operations performed in regional hospitals in upper East Tennessee in 1956

	No.	*Per cent*
Major operations performed	4419	
Operations performed by Board or Board Qualified surgeons—		
General surgeons	1577	36
Sub-specialists	1358	30
Operations performed by general practitioner surgeons	—	—
Operations performed by surgeons	1484	34

The surgical staffs of these four regional hospitals show quite a contrast to those of the small hospitals (table 6). Of the 80 surgeons who operated in 1956, 51 (64 per cent) were Board Diplomates or Board Qualified surgeons, 23 (29 per cent) were general surgeons and 28 (35 per cent) were subspecialists. There were only 29 (36 per cent) general practitioner surgeons.

The number of major procedures performed by these groups of surgeons during 1956 follows closely their incidence (table 7). There were 4419 major operations of which 2935 (66 per cent) were performed by Board Diplomates or Board

TABLE 8

Minor operations performed in regional hospitals in upper East Tennessee in 1956

	No.	Per cent
Minor operations performed	6878	
Operations performed by Board or Board Qualified surgeons—		
General Surgeons	2178	32
Sub-specialists	2188	30
Operations performed by general practitioner surgeons	—	—
Operations performed by surgeons	2512	38

TABLE 9

Regional hospitals—upper East Tennessee (1956)

		Major	Minor
	Per cent	Per cent	Per cent
Board or Board Qualified—			
General surgeons	29	36	32
Sub-specialists	35	30	30
All Board or Board Qualified surgeons	64	66	62
General practitioner surgeons	36	34	38

Qualified surgeons and 1484 (34 per cent) by general practitioner surgeons. There was an advantage in volume for the qualified general surgeon who performed 36 per cent of the major surgery.

There were 6878 minor procedures in these regional hospitals in 1956 of which 4366 (62 per cent) were performed by Board Diplomates or Board Qualified surgeons and 2512 (38 per cent) by general practitioner surgeons (table 8).

Consideration of these data concerning the new regional hospitals in upper East Tennessee supports the conclusion that the majority of the surgeons in these institutions have had basic training experience satisfying the best clinical criteria. This represents a change which has occurred since World War II (table 9). Prior to the last great war medium sized community hospitals were staffed largely by general practitioners who controlled medical policy in their institutions. Since 1946 the picture has been changed radically by two factors: first, the building of larger community hospitals by federal, state and local financial collaboration has given hospital control to boards of directors representing the community rather than the profession. Secondly, the emphasis upon specialty training has provided a sufficient number of properly trained men who have chosen to work in the new, well organized regional hospitals, most of which have staff organization which protects the legitimate specialist in his desire to develop a reasonable volume of practice. The trend in these hospitals is toward the withdrawal of surgical privileges from all but surgical board diplomates; probably another medical generation will see this entirely achieved.

As a corollary to the change in the quality of medical personnel, as based upon criteria of training, there has developed a corresponding improvement of medical services offered through the regional hospitals. Only 20 years ago, patients traveled by train from East Tennessee to Baltimore and Nashville for all but the simpler surgical procedures; later, with changing means of tranportation, Winston-Salem and Durham became popular. At the present time these well known medical centers have few services to offer which cannot be provided in the regional hospitals of our area. The effect of this change has been to distribute good medical care to more people, many of whom could never reach the medical centers.

The contrast between the small hospital and the regional hospital in Tennessee is indicative of a wholesome growth in the scope and quality of medical services. The ideal of equally excellent medical coverage for the whole population must be reached if socialization of medicine is to be avoided. Since the modern practice of medicine is centered about the hospital, it is a logical focus for such efforts. It is obvious that most surgery is performed elsewhere than the medical center, much of it in very small non-accredited local hospitals, and it is imperative that renewed efforts be made to improve the quality of medical services in these areas. Organized medicine should lead out in such a program.

Certain suggestions have been made earlier for the improvement of the service provided by the small hospital. The regional hospital is the bright spot of the present medical care picture, but it too needs continued stimulation to attain its highest possibilities. Although regional hospitals are staffed with well trained surgeons, physicians, roentgenologists, pathologists and anesthesiologists and although they have complements of excellent nursing and laboratory personnel there is lacking an element which cannot be supplied

by these. Excellence in surgical practice at least depends in part upon the development of the surgical team concept. A great need in the new regional hospitals is for well prepared house officers who can give constant care to the critically ill patient since it is becoming increasingly difficult for the responsible surgeon to provide such constant care because of the complexities of modern hospital and private practice routine. The association between staff surgeons and house officers is always a mutually stimulating experience. The younger man acquires the knowledge of the older man, and he in turn profits by the stimulation of teaching. The arrangement reaches its ultimate effectiveness when it can be guided by the expert supervision of medical school faculty staff members.

Certain regional hospitals are now employing on a voluntary basis third and fourth year medical student-interns during the "off-quarters." This is a mutually rewarding experience although the informality of the arrangement denies the privilege of medical faculty supervision. One of our upper East Tennessee regional hospitals, however, invited and obtained the appointment of a medical faculty "Committee on Education" which set up a program of instruction for student-interns which is followed strictly. Other regional hospitals are providing one or two years in surgical residency training particularly for those trainees who need a short general surgical experience before proceeding with surgical specialty training.

It would appear that the necessity which faces the medical school to find more clinical material for its surgical trainees and the need of the regional hospital for house officers to complete its surgical teams and to stimulate its staff surgeons intellectually should find a mutually profitable solution. Such an arrangement would become possible only with the willingness of the medical schools to participate in efforts in education far removed from their walls and with the willingness of the regional hospitals to bear most of the expense. Additional faculty members would be required and administrations of both institutional groups need to be persuaded of the importance of the effort. But the dividends to be gained from such an investment in time, energy and money, if practiced widely, seem to be incalculable. These would be counted as additional facilities for training, and an improved medical service which would bring us closer to the ideal of uniformly excellent medical care for the whole population.

Johnson City
Tennessee

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THE ROLE OF SIMPLE SKIN BIOPSY IN UNCERTAIN INFLAMMATORY CARCINOMA OF THE BREAST

JAMES C. NEELY, M.D.

Cincinnati, Ohio

The clinical picture upon which usually has rested the diagnosis of inflammatory carcinoma of the breast was delineated in 1924 by Lee and Tannenbaum, and reaffirmed in 1938 by Taylor and Meltzer.[5] Largely through their efforts and the work of Haagensen, the condition is categorically considered to be a contraindication to surgery, although a few still advocate radical mastectomy.[6] The prevailing concept rests on the belief that palliative radiation is preferred when no evidence of prolongation of life can be expected from surgery, as has been shown.[4]

During the past seven years at the Cincinnati General Hospital 289 breast carcinomas have been treated. Of these, four were classified as inflammatory. In 7000 cases of malignant lesions of the breast at the Mayo Clinic, 74 (about one per cent) were judged inflammatory.[6] Taylor and Meltzer placed the incidence at four per cent. It is readily apparent from the rarity of the lesion that individual experiences are very meager and that clinical errors will continue to be made as long as diagnosis is based primarily on physical findings. This is even more paradoxical when we realize that the therapy for most breast lesions is based on gross and microscopic pathologic diagnosis while this, the rarest and worst of breast carcinomas, is relegated to physical diagnostic acumen.

We wish to emphasize a point that has not been made clear heretofore, namely that a simple skin biopsy may obviate unwarranted surgery in doubtful cases of inflammatory breast carcinoma.

CASE REPORT

A 57-year-old colored female was first seen in the Surgery Clinic of the Cincinnati General Hospital on November 22, 1957, complaining of progressive painful enlargement of her left breast of three months' duration. A diffuse induration, but no discrete mass was felt, and no discoloration or nipple discharge was recorded. The patient was observed for one week during which time she developed a slight reddish discoloration above the areola. An enlarged axillary lymph node was palpated and biopsy was recommended. The patient refused treatment. She returned to the Clinic two months later and was admitted to the hospital on February 2, 1958, giving no history of breast trauma, weight loss, anorexia, chronic cough, bone pain or possible pregnancy.

Admission physical examination showed an alert, cooperative, uncomplaining patient in no distress. Her temperature was 98.6°, pulse 88, respirations 20, and blood pressure 200/100. The head and neck were negative and the chest was clear to percussion and auscultation. The heart was slightly enlarged but no murmurs were present. A firm, 1-cm. axillary lymph node was described. The left breast was one third again as large as the right, and generalized induration was present. Orange peel edema was present but no reddish discoloration was noted, and tenderness was not conspicuous. The right breast was normal, and the liver, spleen, and kidneys were not palpated. Rectal and pelvic examinations were normal.

The hematocrit was 38 per cent, WBC 4,500 and urinalysis negative. The Kahn test was positive. X-rays of the chest were negative.

The patient was observed for two weeks with the provisional diagnosis of mastitis. She received 500 mg. of Chloromycetin every 6 hours, and during this period she remained essentially afebrile and had no change in leukocyte count. Because of the appearance of a discrete "fist-sized," tender, but non-fluctuant mass a simple mastectomy was carried out.

The pathologic specimen showed tumor cells extending to all margins of the resection, with no dominant tumor mass, but with diffuse tumor involvement of breast parenchyma and dermal lymphatics (fig. 1).

The lesion has been rapidly progressive and the wound has failed to heal. X-ray therapy is being given.

DISCUSSION

The clinical picture of inflammatory carcinoma when completely evolved is classical and not easily forgotten. Haagensen states, in accord with the present case, that signs of breast enlargement, induration, redness, and skin edema were manifest at some time in all 58 cases so classified at the

Presbyterian Hospital from 1915 to 1950. Recorded as presenting symptoms, however, a dominant tumor was present in 67 per cent of the cases, breast pain in 33 per cent, redness in 38 per cent, and enlargement in 33 per cent, while tenderness on pressure, induration, skin edema, increased skin warmth, and all other signs had an incidence of less than 14 per cent.[4] It is obvious therefore that most patients with this disease will not present with the classical complex and that the differential diagnosis must include such conditions as Paget's disease, tuberculosis, erysipelas, chronic inflammation, radiation dermatitis, abscess, ductal ectasia, necrosis of carcinoma, carcinoma en cuirasse, lymphoma, sarcoma, and even noxious skin irritants.

The difficulty in mistaken differential diagnosis was described an early as 1911 by Schumann who incised an "inflamed cancer" for an abscess in a lactating breast which failed to heal. Da Costa in 1919 advocated incisional biopsy "in any case of mastitis persisting longer than two weeks" and stated that the diagnosis of cancer should be strongly entertained. Taylor and Meltzer reemphasized this in 1938,[8] and of Donnelly's five cases reported in 1948[3] three diagnoses were made on the basis of skin biopsy. Despite this background the impression is that the diagnosis of inflammatory carcinoma most often rests on clinical findings and cases are still reported in which only after an attempted incision and drainage fails to heal is the lesion suspected.[4] In such cases the parenchymal carcinoma is diffuse and no mass is encountered for biopsy; sections of skin either may not be submitted or the pathologist may not be alerted to their possible significance. At the Cincinnati General Hospital special skin sections are done routinely on all breast specimens.

Although it is well recognized that all varieties of breast carcinoma may become inflammatory the *sine qua non* is not a clinical picture but the histopathologic finding of emboli of tumor cells in the dermal and subepidermal lymphatics (fig. 1).[5, 8] Chris emphasizes the distinction between primary inflammatory changes which appear simultaneously with the tumor and a secondary type of inflammation which is seen when occult underlying carcinoma has been present for a time and then spreads. In either event it is obvious that the tumor is of the diffuse rapidly spreading type and may be missed except by appropriate skin examination.

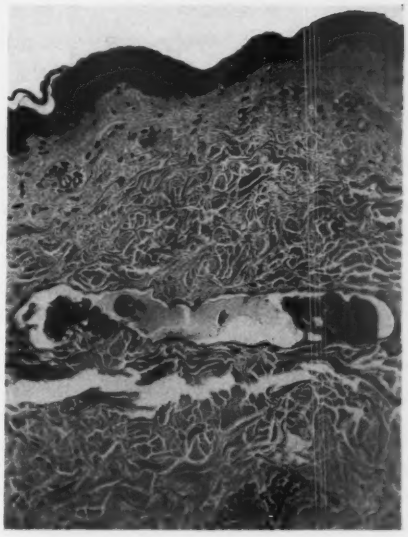

FIG. 1. Photomicrograph of skin sections from a case with inflammatory carcinoma of the breast. Note emboli of tumor cells in dilated dermal and subepidermal lymphatics. Edema and chronic inflammatory reaction are also present. × 96.

Finally, and most noteworthy, skin biopsy properly timed and placed will not only hasten diagnosis but will preserve the tissue mass desirable for radiotherapy, and reduce morbidity.

SUMMARY

Inflammatory carcinoma of the breast, the most malignant of breast carcinomas, is the only breast lesion often diagnosed exclusively on clinical grounds. Because of its rarity few clinicians can be conversant enough to deal with it on this basis, and surgical errors of commission continue to be made.

A case is reported where simple mastectomy was done inadvisedly for an unresolving mastitis. The pathological diagnosis was inflammatory carcinoma.

The rationale and advantages of simple skin biopsy are discussed in cases of questionable inflammatory breast carcinoma. It is shown how this approach can hasten the diagnosis and disposition of these cases.

Department of Surgery
Cincinnati General Hospital
Cincinnati, Ohio

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CAROTID CAVERNOUS FISTULAS: DIAGNOSIS AND SURGICAL MANAGEMENT*

LT. COL. GEORGE J. HAYES, M.C., U.S.A.

Washington, District of Columbia

A carotid cavernous fistula consists of an opening in the carotid artery as this artery passes through the cavernous sinus (fig. 1). The fistula allows arterial blood to enter the extradural venous circulation at the base of the skull which causes an increase of pressure in this system as well as a reversal of the usual venous drainage path from the orbit to the cavernous sinus. The majority of reported cases have followed head injury, but spontaneous onset is not infrequent. Even following head injury, this complication is rare.

When this condition is recognized soon after its onset and promptly treated, the results are gratifying to both the patient and his surgeon. If the condition is not diagnosed and treated, it is, at best, distressing to the patient. It may seriously impair function, or even constitute a threat to his life.

The exhaustive reviews of Sattler,[5] Dandy,[2] and Hamby,[3] on this subject, give in detail the historical and minute anatomic features of this lesion. The purpose of this brief presentation is to draw attention to this entity so that recognition will bring the patient to early treatment. Also another mode of therapy† is outlined and an explanation offered for the failures which can occur even though the treatment has apparently been complete.

The relation between "pulsating exophthalmos" and carotid cavernous fistula was recognized by Travers[7] as early as 1809. Hamby states, "An individual operator sees so few of these cases that he does not have the opportunity to accumulate any worthwhile series of them." Although this is true, collection and analysis of reported cases have established pounding headache, exophthalmos—usually of a pulsating type—chemosis, diminished visual acuity, and paralysis of one or more of the nerves serving the ocular muscles as the principal clinical features. Tables 1 and 2 show the distribution of these findings in the 14 patients who form the basis for this discussion.

The universal complaint of all these patients was a pounding or throbbing headache. No patient spontaneously mentioned the noise of the bruit, but when questioned, all admitted hearing it. Exploration of this phenomenon revealed that the terms "pounding" or "throbbing" were meant by the patients to include the quality of noise as well as that of pain. Hence, all patients should be questioned specifically after head injury regarding this symptom. Auscultation of the orbits should also be part of the initial and final physical examinations, regardless of the severity of the injury.

The degree of exophthalmos had little relation to the size of the fistula. In general, it was greater the longer the fistula had been present. Combinations of cranial nerve malfunction were seen but are not considered of specific diagnostic value. Systemic vascular effects were not observed.

Travers, who made the first diagnosis of this condition, was also the first to attempt to cure it by surgery. He ligated the common carotid artery and cured his patient. Since his time, the surgical attack has been directed primarily toward this artery or its major branches, singly or in combination. A number of large groups of patients treated in these ways have been collected and analyzed from time to time. Although there are variations from group to group, it generally can be stated that a 50 per cent chance of cure can be expected after such surgery. Recurrence, however, is common (10 to 20 per cent), hemiplegia frequent (20 per cent), and the mortality risk is from 5 to 10 per cent.

* From the Neurosurgical Service, Walter Reed Army Hospital, Walter Reed Army Medical Center, Washington 12, D. C.

† Since preparation and presentation of this report, an article not contained in the Cumulative Index Medicus has been brought to my attention by Dr. Rudolph Jaeger,[10] who first used this mode of treatment in 1942. Dr. W. Keasley Welch[8] has informed me that he has used this method of treatment and knows that at least two other surgeons, Dr. James Galbraith and Dr. Carl Graf, have also used it.

Presented during the Baltimore Assembly of The Southeastern Surgical Congress, March 10–13, 1958, Baltimore, Maryland.

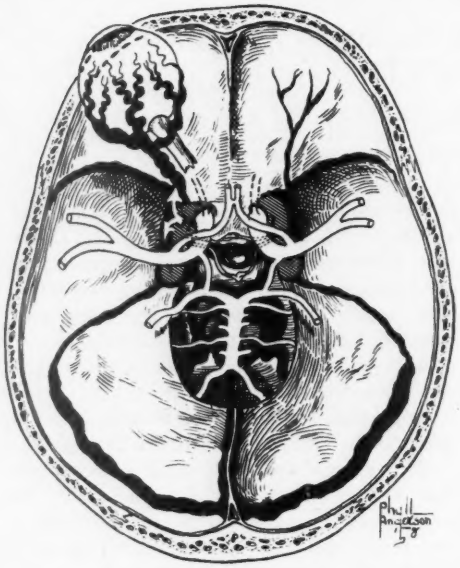

FIG. 1. Schema of carotid cavernous fistula

In an effort to increase the certainty of closure of the fistula, Zeller[9] occluded the internal carotid intracranially after ligation in the neck. This isolates the fistula from its major blood supply, particularly if the ophthalmic artery is occluded. The first step (carotid ligation) in this approach, however, carries the same risks as the older and more classical method.

As a result of these deficiencies in treatment, the problem was examined to determine whether a better surgical approach could be devised. The first step was to review the blood flow pattern of the circle of Willis. Anatomic studies have pointed out many variations from the norm in this structure, but clinical experience has shown that in patients who come to surgical consideration, the circle is almost always a functioning structure. The following argument is based on the hemodynamics of the "normal" circle of Willis.

Ordinarily, the pressure relationships in the circle of Willis (fig. 2) are such that very little cross circulation occurs from side to side in the anterior portion of the circle. Neither is there much exchange between the carotid and vertebral systems. When a reduction of pressure occurs in one of the carotids or vertebrals, however, the posterior and anterior communicating arteries

TABLE 1

Incidence of clinical signs and symptoms of carotid cavernous fistula

Total patients	14
Headache	14
Bruit	14
Engorgement of retinal veins	14
Exophthalmos	10
Unilateral pulsating	8
Bilateral Nonpulsating	2
Chemosis	4

TABLE 2

Cranial nerve abnormalities caused by carotid cavernous fistula

Cranial Nerves					
II		III	V		VI
Diminished visual acuity	Field change	Partial or total paralysis	Symptoms of pain	Hypalgesia 1st or 2nd division	Paralysis
2	2	7	14	4	8 (1 bilateral)

enable blood to be shunted into the threatened area of the brain.

Angiographic studies have shown that there are two general types of carotid cavernous fistulas (fig. 3)—those which allow escape of all or practically all of the blood flow from the torn carotid into the cavernous sinus, and those (fig. 4) which allow a significant flow of blood to the brain, despite the fistulous opening into the cavernous sinus. If a patient falls into the first group and he does not become hemiplegic immediately upon formation of his fistula, his circle of Willis is evidently competent to supply blood to the anterior cerebral and middle cerebral branches of the torn carotid which is no longer supplying the brain with blood.

If this internal carotid is occluded in the neck (fig. 5) and the fistula remains open, then the total blood loss suffered by the brain is the summation of one internal carotid supply plus whatever continues to go out through the fistula. This latter amount is provided by the other three vessels *via* the circle of Willis. Such loss may be enough to cause hemiplegia or death as a result of cerebral ischemia.

If a small fistula allows only partial loss of

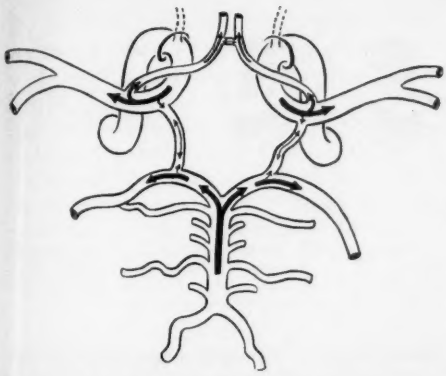

FIG. 2. Arrows show major blood flow pattern in circle of Willis.

FIG. 3. Angiogram showing that blood is totally diverted into the cavernous sinus.

atrerial blood into the venous system, the brain, to some extent, may still be dependent upon that internal carotid artery for its blood supply. In addition, there is no reliable clinical information regarding the competency of the circle of Willis. If the internal carotid artery is abruptly ligated in the neck, the circle of Willis may not be able to compensate promptly for the loss of this blood. Hemiplegia or death may then follow primary intervention in the neck.

As a result of these considerations, the following approaches in the surgical treatment of carotid cavernous fistulas are proposed. All patients with carotid cavernous fistulas should be subjected to carotid angiography, at least on the involved side, and preferably bilaterally. This procedure enables the surgeon to classify a particular patient into the "total diversion" group or into the "partial diversion" group. If the patient belongs in the total diversion group and is not hemiplegic, the competency of the circle of Willis is without question. The first operative step (fig. 6) for this type of patient is occlusion of the intracranial portion of the internal carotid artery at a point between the ophthalmic artery and the posterior communicating artery. Next, the ophthalmic artery should be closed as it arises from the internal carotid artery, but it is not possible to do this in every instance. Then, during the same operative session, the cervical portion of the internal carotid is doubly ligated and sectioned. The brain, during this procedure, is at no time deprived of more blood than it has been losing into the fistula.

In those patients in whom only partial diver-

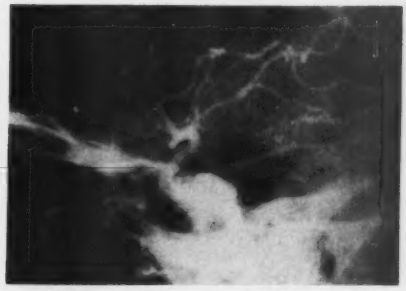

FIG. 4. Angiogram showing partial diversion of blood into the cavernous sinus.

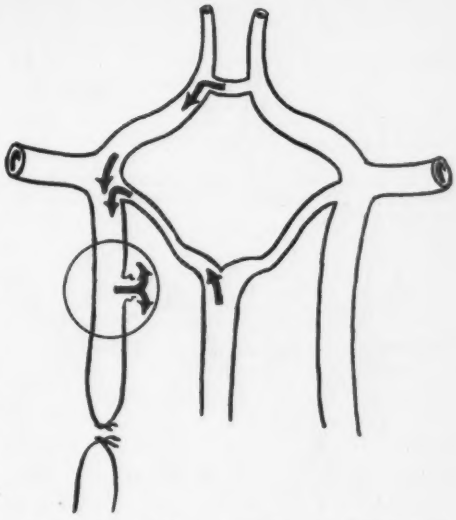

FIG. 5. Loss of blood from the circle of Willis if the fistula remains open following ligation of internal carotid.

sion of blood occurs, our present knowledge indicates that we should endeavor to give the circle of Willis an opportunity to demonstrate that it can substitute for the loss of that blood which the affected artery is still supplying the brain. Therefore, a somewhat different approach is used (fig. 7). The first step is to expose the internal carotid artery in the neck and to apply an adjustable clamp, such as Dr. Selverstone's,[6] to the artery and to close the clamp so that the artery is almost, but not quite, completely occluded. Following this, the patient is observed carefully for signs of cerebral vascular insufficiency. Should any such signs develop, the clamp is opened; then, over a period of days, gradually brought back to the point of almost complete closure. It is never completely closed. Such occlusion of the artery is maintained for at least a week. This approach accomplishes two purposes. First, the circle of Willis is, in effect, given a stress test which demonstrates its ability to provide the brain with an adequate supply of blood. Secondly, the cerebral vascular circulation is enabled to adjust gradually to loss of blood from the involved carotid artery. After this period of test and adjustment, the operative steps are the same as those used in the patients with total diversion. In one operative session, intracranial occlusion of the carotid, followed by ligation and section of the cervical portion of the carotid artery is performed.

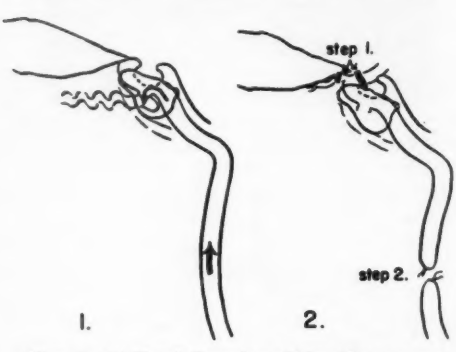

FIG. 6. (1) Total diversion of blood into cavernous sinus; (2) sequence of occlusion of internal carotid artery.

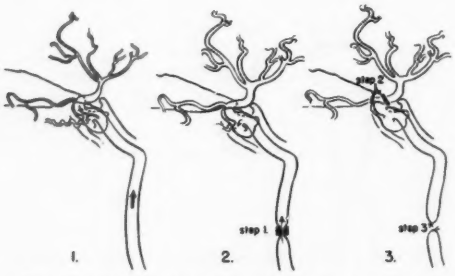

FIG. 7. (1) Partial diversion of blood into cavernous sinus; (2) subtotal occlusion preliminary to definitive surgery; (3) sequence of occlusion of internal carotid artery.

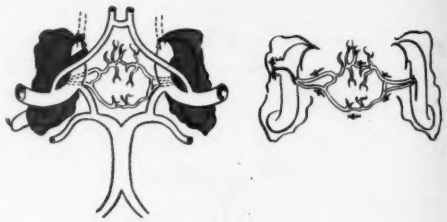

FIG. 8. (*Left*) Small vessels leaving carotid artery as it passes through cavernous sinus (shaded); (*right*) possible channels for maintenance of fistula despite occlusion of carotid.

RESULTS

Eight of the patients discussed in this series were treated by primary cervical internal carotid artery ligation with subsequent intracranial carotid occlusion. Four of these patients were in the total diversion group and four had partial diversion of carotid flow. In no patient did hemiplegia develop, nor were there any deaths. The symptoms caused by the fistula itself were relieved in all of the patients, although one had a bruit audible to the examiner only. The last six patients treated by me have been managed by the primary intracranial approach previously discussed. Four of these had total diversion of blood into the fistula; two had partial diversion. There have been no deaths, nor has hemiplegia occurred. One patient has persistent abducens nerve weakness; also, one patient in this group has a bruit which can be heard only by the examiner.

From these results, there appears to be little to support the contention that the newer method of treatment is superior. The theoretic considerations, however, suggest that as larger series of patients are treated, the risks of hemiplegia, recurrence, and death will be diminished.

The existence of a bruit, despite apparent isolation of the fistula from the arterial circulation, demands an explanation (fig. 8). There are several small arteries arising from each internal carotid artery as it passes through the cavernous sinus.

The branches enter the pituitary fossa and anastomose within it. If correction of a carotid cavernous fistula is not accomplished soon after its onset, it is reasonable to suppose that these anastomosing vessels could develop a circulation great enough to maintain the fistula. If this blood flow through the fistula causes continued signs and symptoms, introduction of muscle into the carotid artery, as described by Brooks[1] or Hamby and Gardner,[4] might be necessary.

SUMMARY

A carotid cavernous fistula may follow even a trivial head injury or it may be spontaneous in onset. Diagnosis is usually simple if the examiner elicits a history of unilateral headache of a pounding or throbbing nature, with or without pulsating exophthalmos, and if he can hear a bruit on auscultation of the orbits. Angiographic demonstration of blood flow pattern at the cavernous sinus shows that there are two types of fistulas—those in which all blood from the internal carotid is diverted into the cavernous sinus and those in which a significant supply of blood still reaches the brain, despite the fistula.

The results achieved after primary ligation of the internal carotid artery in the neck followed by intracranial occlusion of the internal carotid have been described. An additional surgical technique has been outlined, and the results in six instances reported. In this approach the internal carotid artery is first occluded intracranially and then in the neck. The precise steps of this method are guided by the amount of blood escaping through the fistula.

An explanation is offered for the persistence of a fistula when the involved segment of artery presumably has been isolated from the general circulation.

Walter Reed Army Medical Center
Walter Reed Army Hospital
Washington, D. C.

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GASTRIC SARCOMA

COLEMAN C. JOHNSTON, M.D., F.A.C.S.

Lexington, Kentucky

Gastric sarcoma is an unusual tumor. Snoddy[1] in 1952 reviewed the world literature to find only 474 cases reported. Ewing[2] in the early 1920's estimated that gastric sarcoma accounted for about 1 per cent of all gastric malignancies. By 1950, Marshall and Meissner[3] on the other hand, reported a 3.5 per cent incidence of sarcoma in a review of 41 cases found among 1711 gastric malignancies seen at the Lahey Clinic from 1929 to 1949. Other figures would tend to substantiate this increasing incidence of gastric sarcoma which is probably relative as well as actual.

Gastric lymphosarcoma, comprising about 60 per cent of these tumors, was first described by Morgagni[4] in 1751, and surgically treated by Virchow[5] in 1887.

The first gastric leiomyosarcoma was described by Virchow[5] in 1862. Although leiomyosarcomas comprise between 10 and 20 per cent of gastric sarcomas, only 40 such tumors were found at the Mayo Clinic between 1907 and 1946. Walters[6] has commented that one leiomyosarcoma could be expected for every 1000 gastric carcinomas.

The fibrosarcoma and neurosarcoma are even more unusual, while only two liposarcomas[2] of the stomach have been reported in the literature.

Among 66,041 admissions to the St. Joseph Hospital in Lexington, Kentucky, during the past six years, there have been only six gastric sarcomas. We have treated four of these lesions and because of the profound influence the various characteristics of these tumors have on the diagnosis, treatment, and prognosis of the disease, it would seem appropriate at this time to discuss some of the important features observed during this limited experience.

PATHOLOGY

Ewing defines sarcoma as a malignant tumor composed of cells of connective tissue origin. There have been devised many different classifications of sarcoma most of which seem only to add confusion to a still controversial subject. For the sake of simplicity the following will serve in presenting the subject of gastric sarcoma.

Sarcoma of Connective Tissue Origin

1. Leiomyocarcoma
2. Fibrosarcoma
3. Neurofibrosarcoma
4. Liposarcoma

Sarcoma of Lymphoid Tissue Origin

1. Lymphocytoma
2. Lymphoblastoma
3. Reticulum Cell Sarcoma
4. Giant Follicular Cell Sarcoma
5. Hodgkins Sarcoma

Mallory[7] holds that sarcoma must develop as an aberration of one of the 15 original body cells. Hence, the fibrosarcoma arises from the embryonic fibroblast, etc. He divides the lymphosarcoma into two groups, those arising from the reticulum cell and those from the lymphoblast.

Nunn[8] is inclined to believe the various forms of lymphosarcoma, commonly termed the malignant lymphoma, are simply different manifestations of the same or a very closely related pathologic process, arising from the primary lymphoblast found in the germinal center of the lymph node. Custer and Bernhard[9] are in accord with this thesis and have shown through serial biopsy studies that the lymphocytic type, the follicular types, the reticulum cell sarcoma and the Hodgkins lymphoma are all transitory phases of a single basic neoplastic process.

Migliaccio[10] contends that these solitary tumors in the gastrointestinal tract are in reality only local manifestation of a systemic disease, however the surgical cure or long term survival without systemic manifestations would tend to lend doubt to this concept. Sugarbaker and Craver[11] explain this phenomena by claiming that lymphosarcoma may be a unicentric or multicentric disease.

Both the connective tissue sarcoma and the malignant lymphoma originate in the submucosal and subserosal tissues of the stomach. The leiomyosarcoma often grows to tremendous proportions, either extending into the lumen or

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protruding outward from the viscus. Mucosal ulceration is a late manifestation, resulting from pressure necrosis, or compromise of the local blood supply through overgrowth of tumor tissue with resultant areas of focal necrosis.

The malignant lymphomas also growing within the visceral wall, infiltrate the muscular coat, often destroying the muscle and its nerve supply. They have a delicate reticular structure and therefore tend toward infiltrative rather than constrictive alteration of the viscus. They range all the way from extensive flattened intramural lesions to large fungating tumors, occupying much of the visceral lumen. Again in this group ulceration and necrosis are late manifestations.

With time both lesions may become large nodular or fungating growths with areas of ulceration or deep sinus formation. They usually protrude into the visceral lumen. Grossly the tissue is firm and rubbery but not stony hard as in carcinoma. The nodular lesions may be single or multiple. Usually the base is circumscribed in a limited area and therefore both the leiomyosarcoma and the lymphosarcoma are amenable to a less extensive resection than is carcinoma of the stomach.

Metastasis in both types of sarcoma occur late in the disease. They may be local or distal and either lymphatic or blood borne. Marked local glandular enlargement on a purely inflammatory basis is not uncommon and often deceptive, after necrosis or ulceration have supervened.

CASE REPORTS

Case 1. Mrs. K. G., a 51-year-old white school teacher, was seen on May 12, 1952. She had a history of weight loss and persistent and recently progressive indigestion of about a year's duration. Upper gastrointestinal x-ray series seven months prior to admission was reported as negative. A recent study revealed a small crater midway of the lesser curvature (fig. 1). There was nothing to suggest malignancy. Gastroscopy revealed an area of mucosal thickening with a granular surface and a loss of pliability and motility along the midportion of the lesser curvature. These findings were suggestive of an infiltrative process obviously implying malignancy (fig. 2). The area was easily traumatized during examination, but no actual crater could be seen.

Physical examination was essentially negative. There was mild discomfort on gentle pressure over the epigastrium, but no palpable mass. Laboratory findings were all within normal limits. Exploratory laparotomy revealed an area of induration and thickening along the mid-portion of the lesser curvature of the stomach, with translucent lymph glands along the greater and lesser curvatures, ranging from 3 to 10 mm. in diameter. Gastrotomy revealed no crater but a thickened, granular appearance of the mucosa along the lesser curvature.

Biopsy of the mucosa and several adjacent glands presented for frozen section were interpreted as no more than inflammatory changes. About three-fourths of the stomach was resected (fig. 3). On gross examination there was a diffuse thickening and firmness of the gastric wall, and our pathologist felt the possibility of a lymphoid tissue tumor, undiagnosed at frozen section, could not be ruled out. Permanent section established a pathologic diagnosis of lymphosarcoma, without involvement of the adjacent lymph glands (fig. 4). In this instance gross examination and palpation suggested a lymphoid tissue tumor not diagnosed at frozen section, although confirmed on permanent pathologic study.

This patient had an uneventful postoperative course and has remained symptom free without evidence of local or systemic disease for five and one-half years.

Case 2. W. S., A 52-year-old colored farmer, was admitted on June 5, 1952 with a history of repeated episodes during the past two years of indigestion which was relieved by soda or "black draught." There was soreness and deep discomfort in the epigastrium with gaseous eructation accentuated by nervousness and associated with occasional episodes of vomiting.

Physical examination revealed a well developed and well nourished colored male in moderate discomfort, complaining of mild epigastric pain. The abdomen was soft with tenderness elicited on gentle pressure over the epigastrium. No mass was palpable. No other abnormalities were noted. Laboratory findings were essentially negative. X-ray examination of the stomach and duodenum revealed what was thought to be a duodenal ulcer, however irritability and narrowing of the prepyloric area suggested the possibility of a gastric lesion (see fig. 5). Gastroscopic examination revealed a normal mucosal pattern throughout the upper two-thirds of the stomach, however antral spasm prevented visualization of the antrum and pylorus. "Coffee-ground" material could be seen to regurgitate proximal to the antrum, indicating distal pathology although the lesion itself could not be seen.

Exploration revealed a rubbery, thickened, subserosal lesion about 8 cm. in length, involving the antrum, pylorus and the first portion of the duo-

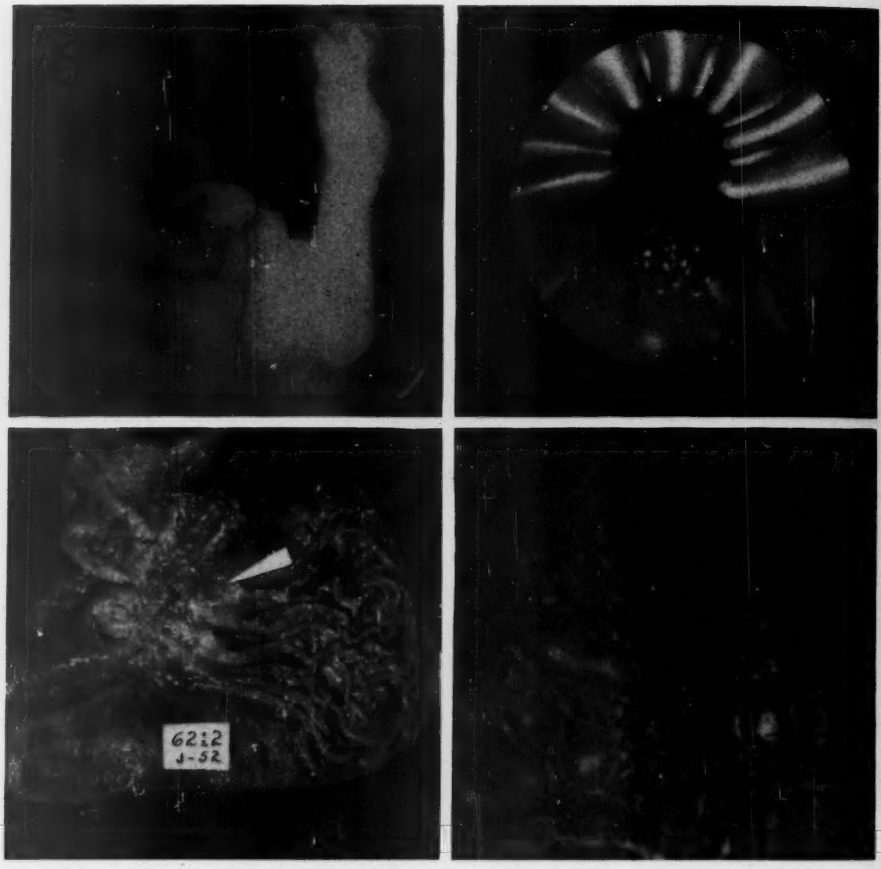

FIG. 1 (*Top left*). X-ray revealed a small shallow crater midway of the lesser curvature with nothing to suggest malignancy.

FIG. 2. (*Top right*). Gastroscopy revealed an area of mucosal thickening with a granular surface and a loss of pliability and motility along the mid-portion of the lesser curvature, suggesting an infiltrative process implying malignancy.

FIG. 3 (*Bottom left*). The resected specimen revealed a limited area of induration and thickening involving the mid-portion of the lesser curvature.

FIG. 4 (*Bottom right*). Microscopic examination revealed a round cell infiltration of the gastric mucosa and muscularis characteristic of the malignant lymphocytes found in the reticulum cell sarcoma.

denum. There were many large glands above and below the tumor, extending well up to the lesser curvature, grossly suggesting inflammatory changes. Frozen section of a large node was reported as negative for metastatic disease. Resection of the lower two-thirds of the stomach, first portion of the duodenum and all gland-bearing tissues was done with an anterior Hofmeister anastomosis (fig. 6). The postoperative course was uneventful.

On permanent section this lesion proved to be a reticulum cell sarcoma of the stomach and duodenum with metastatic involvement (figs. 7 and 8). Although this lesion involved both the stomach and duodenum, the incidence of gastric sarcoma is considerably higher than is duodenal sarcoma.

About two months after operation, the patient returned complaining of deep epigastric pain and tenderness. There was moderate weight loss but no palpable tumor. Having established a diagnosis of reticulum cell sarcoma, it was quite evident that if this patient should develop symptoms suggesting a local recurrence, x-ray treatment would be indicated. A course of deep x-ray was given. The symptoms subsided. This patient has sur-

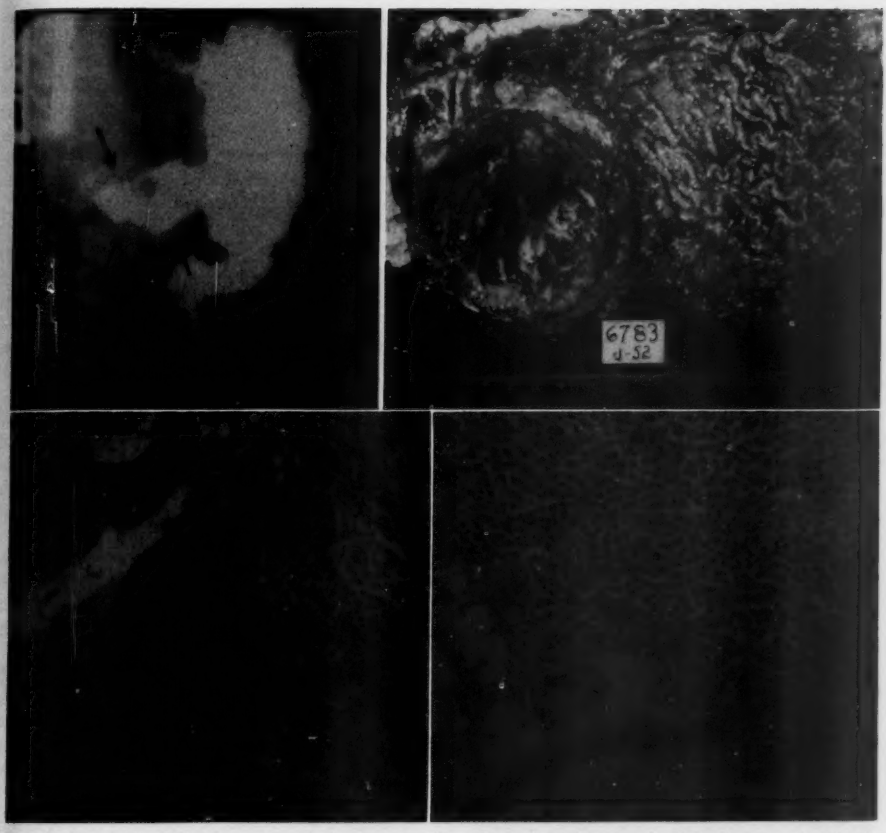

FIG. 5 (*Top left*). G.I. x-ray revealed what was thought to be a duodenal ulcer, however irritability and narrowing of the prepyloric area suggested the possibility of a gastric lesion.

FIG. 6 (*Top right*). Exploration revealed a rubbery thickened subserosal lesion about eight centimeters in length, involving the antrum, pylorus and first portion of the duodenum.

FIG. 7 (*Bottom left*). Microscopic study, as in case 1, revealed the lesion to be a reticulum cell sarcoma invading the gastric mucosa.

FIG. 8 (*Bottom right*). This section reveals the malignant lymphocytes invading Brunner's glands of the duodenum.

vived five years and a recent examination reveals no evidence of malignant disease. This case illustrates the value of x-ray therapy in conjunction with operation in treating malignant lymphoma.

Case 3. D. S., a 43-year-old housewife, was seen in consultation on April 17, 1951 for gastroscopic examination because of a 12-year history of tarry stools and occasional hematemesis; she had had 50 transfusions during this period.

Esophagoscopy and x-ray examinations were negative (fig. 9). At gastroscopy a smooth, rounded, submucosal tumor 4 cm. in diameter presented in the mid-portion of the stomach (fig. 10). During the gastroscopic examination fresh blood could be seen to drop from the tumor at about the rate of the patient's pulse. A diagnosis of mesenchymal tumor with mucosal ulceration—probably a leiomyoma—was made.

At exploration by the referring surgeon a smooth, rounded, submucosal, walnut-size tumor presented with an ulcer crater on its distal surface. A generous sleeve resection was carried out and the patient had an uneventful postoperative course.

Pathologic examination revealed the lesion to be a leiomyosarcoma with mucosal ulceration and a deep sinus formation, the base of which was limited to a relatively small area (fig. 11).

Lahey and Colcock[12] and Collins and Collins[13] feel that the leiomyoma is a premalignant lesion.

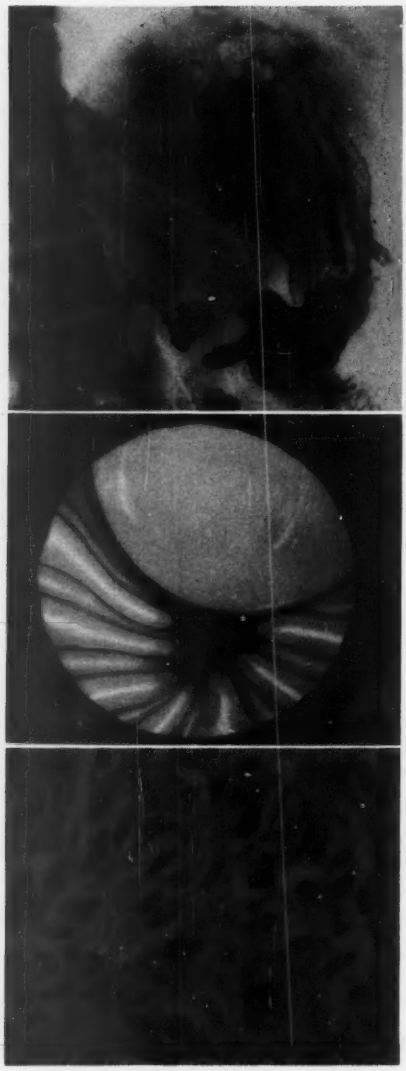

FIG. 9 (*Top*). X-ray revealed no evidence of a golf-ball size tumor in the fundus of the stomach.

FIG. 10 (*Center*). Gastroscopic examination revealed a smooth rounded golf-ball size tumor in the fundus of the stomach, from which blood could be seen to drop at about the rate of the patient's pulse.

FIG. 11 (*Bottom*). The tumor was composed of elongated spindle shaped cells characteristic of leiomyosarcoma and was considered a Grade I lesion. Grossly it was a firm round tumor with mucosal ulceration and deep sinus formation.

At autopsy a search for benign tumors by Rieniets[14] and Dudley and associates[15] reveals a 15 to 20 per cent incidence of leiomyoma. The x-ray diagnosis of isolated tumors in the body of the stomach is often difficult and gastroscopic examination is not infallible, however this case does serve to emphasize the importance of gastroscopic examination in those patients with gastric symptoms and negative x-ray findings.

Case 4. H. P., a 73-year-old colored farmer, was admitted on July 26, 1951 with a six-month history of episodes of mild-to-moderately severe, sharp, colicky pain in the epigastrium. During the three years prior to this he had noticed persistent, annoying epigastric distention. There had been moderate weight loss with anoxemia during the four weeks prior to admission.

Physical examination revealed an alert, well developed but thin, colored man of 73. The findings were essentially negative, except for moderate tenderness on gentle pressure over the epigastrium by the upper abdominal muscles. There were no palpable mass.

Except for a mild secondary anemia with a red cell count of 3.7 million, a hemoglobin of 71 per cent, 27 degrees total and no free hydrochloric acid, the laboratory findings were essentially negative.

X-ray revealed a large tumor in the body of the stomach (fig. 12) and at gastroscopy we observed a large irregular tumor which bled easily during the examination. The possibility of a sarcoma was recorded at gastroscopy because of the odd size, shape and irregularity of the tumor.

Exploration revealed a large intragastric mass in the upper half of the stomach. Frozen section examination of enlarged paragastric glands revealed no evidence of malignancy. Direct biopsy of the tumor was not done. The lesion was believed to be an extensive carcinoma, therefore a total gastrectomy was performed. The patient developed evidence of sepsis and died ten days thereafter.

Pathologic examination revealed a large fungating tumor and also a smaller lesion, each involving a limited area of the stomach wall with limited local infiltration (fig. 13). Microscopic examination of each tumor revealed a spindle cell sarcoma of low grade malignancy (fig. 14).

The importance of presenting this case, though tragic in outcome, rests upon the error in judgement in not establishing a diagnosis by means of direct biopsy of the tumor itself with frozen section of the specimen. Doubtless this is not an uncommon error and may well account in part for the reputed low incidence of gastric sarcoma

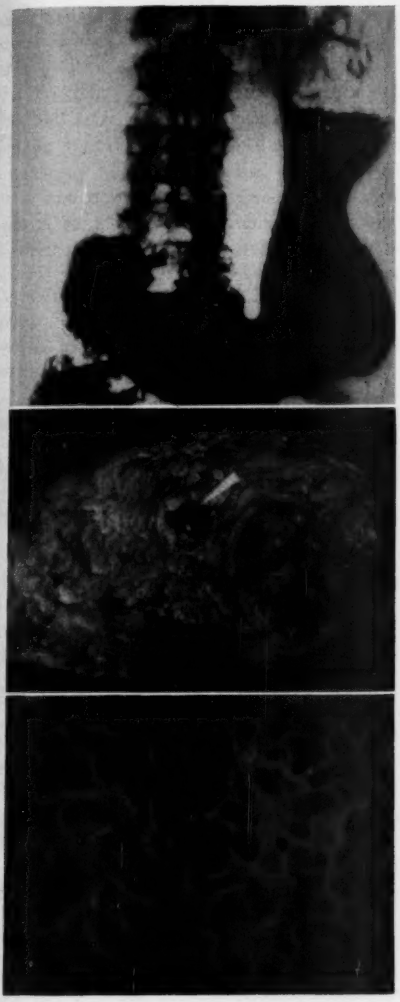

FIG. 12 (*Top*). X-ray revealed a tumor of the fundus of the stomach thought to be malignant.

FIG. 13 (*Center*). Total gastrectomy and splenectomy revealed one large degenerated necrotic tumor in the fundus of the stomach. Distal to this a second small tumor is marked by the pointer.

FIG. 14 (*Bottom*). Microscopic study of each of these tumors revealed somewhat irregular elongated spindle shaped cells characteristic of leiomyosarcoma. This greater irregularity and an increase in mytotic figures over the previous lesion was interpreted as a sarcoma of Grade II malignancy.

among the many large tumors considered inoperable prior to the days when gastrotomy and primary biopsy was believed to be the procedure of choice. Certainly in the light of our present experience an accurate biopsy study would have led to a more conservative approach to the problem with an easier and safer resection, carrying a lower mortality. Again more attention should have been given our gastroscopic findings, for although this was the first in our experience, it has been observed that unusual and bizarre findings in a gastric tumor should always alert the gastroscopist to the possibility of sarcoma. In the absence of extragastric malignancy, the conservative approach might well have eradicated all trace of this localized process.

DISCUSSION

In this study of four cases of gastric sarcoma the average age was 57, or between 7 and 14 years higher than that reported by Brodus or Walters. There are no outstanding clinical characteristics of the disease which would tend to distinguish it from gastric carcinoma. Although many authors have suggested minor differential points, it becomes quite evident that a specific preoperative diagnosis of sarcoma of the stomach made by x-ray or gastroscopy is most unlikely.

At x-ray examination the first of these sarcomas was described as a benign gastric ulcer; the second, a duodenal ulcer with possible antral pathology; the third, (a golf-ball size tumor) was not even seen; and the fourth was reported as a carcinoma of the stomach. All four of these patients were gastroscoped. The first lesion presented a thickened, granular area in the mucosa with loss of pliability and motility, suggesting an infiltrative lesion demanding surgery, instead of benign gastric ulcer as diagnosed by x-ray. In the second tumor, involving both the stomach and duodenum, the lesion could not be seen at gastroscopy because of muscular spasm proximal thereto. The third lesion, a golf-ball size tumor overlooked at x-ray, was readily visualized at gastroscopy and diagnosed a leiomyoma. In the fourth case, gastroscopy revealed a large, irregular tumor (in the fundus of the stomach) which bled readily. The possibility of sarcoma was considered because of the size, shape and unusual character of the tumor. In short then, x-ray overlooked one lesion, diagnosed two as benign, and one as a malignant tumor. Gastroscopy overlooked one lesion, diagnosed two as malignant, and in one a diagnosis of leiomyoma instead of leiomyosarcoma was made. This study again revealed the limitations of x-ray

examination in the diagnosis of gastric diseases. It also emphasizes the inconstant value of gastroscopy alone and in combination with x-ray, as a diagnostic aid; however, these examinations together will supplement one another to great advantage.

Frozen section of the tumor and glands in the first case proved unreliable, and in the second instance grossly involved glands were reported as negative. The third case was not studied thus, while in the fourth, frozen section of an enlarged gland was reported as benign. The tumor itself was not biopsied to the distinct disadvantage of the patient and the clinician. An accurate diagnosis of sarcoma would in this instance have guided the operator toward conservatism and a less radical procedure would have likely ended in a more gratifying result. A frozen section of primary tumor tissue should, therefore, always be done, realizing, however, the limitations of this technique.

Two of the four patients with gastric sarcoma have survived five years or more; the third case cannot be traced for a follow-up; the fourth had no evidence of extrinsic malignancy at operation or autopsy. The second patient had metastatic glandular involvement at operation with presumptive recurrence controlled by x-ray therapy and is now living and well after five years. Marshall records a 67 per cent and 26 per cent five-year survival in leiomyosarcoma and lymphosarcoma respectively. In short, the prognosis of gastric sarcoma is considerably better than that of carcinoma of the stomach. Because of their tendency to remain localized and metastasize late, these lesions will be more readily controlled by conservative surgery than will carcinoma of the stomach.

SUMMARY

The subject of gastric sarcoma is reviewed and four cases of gastric sarcoma are presented. The likelihood of an accurate preoperative diagnosis is small. We are impressed with the limitation of x-ray diagnosis in gastric disease; gastroscopy also is fraught with many shortcomings. Both the roentgenologist and gastroscopist should work in close cooperation with one another. The gross pathology of these lesions may, if studied carefully, suggest the proper diagnosis, whereas frozen section is not always a reliable means of establishing an accurate diagnosis. Direct tumor biopsy is essential; if possible an accurate evaluation of the tumor should be made because less radical surgery will be necessary for cure in gastric sarcoma than in gastric carcinoma. Two of the four patients discussed are alive and well after five years, one cannot be traced, and the fourth had no extrinsic disease at operation or autopsy.

Deep x-ray treatment in the presence of inoperable or recurrent lymphosarcoma of the stomach is of value and, therefore, it is urged that all gastric tumors—however extensive—be explored and biopsied.

211 West Second Street
Lexington, Kentucky

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POSTOPERATIVE DIFFICULTIES FOLLOWING SUBTOTAL GASTRECTOMY

ALBERT L. EVANS, M.D., OLIN S. COFER, M.D., HUGH H. GREGORY, M.D., AND GUY L. CALK, M.D.

Atlanta, Georgia

Although new procedures are constantly being developed and tried, at the present time subtotal gastrectomy remains the operation of choice for the majority of surgeons in the treatment of peptic ulceration. This operation is popular because the mortality is now down around three per cent and because 85 to 90 per cent of the patients are either cured or improved after the procedure.

We agree with Maingot that the procedure of choice for subtotal gastrectomy is the anterior Hofmeister-Polya technique with removal of 75 per cent of the stomach (fig. 1). The afferent loop, usually about 20 cm. in length, is placed at the lesser curvature of the stomach and is isoperistaltic. We abandoned the retrocolic method some years ago because complications about the anastomosis present such a problem. If difficulties should occur following the antecolic method, they can be much more easily remedied. The stomach is amputated in an oblique manner which creates a funnel-like gastric pouch emptying its contents directly into the efferent loop of the jejunum and very little if any of the feeding will enter the afferent loop. The stoma is placed at the greater curvature about 5 cm. in width. Just prior to complete closure of the anastomosis a finger should be passed through both stomas to determine their patency. The jejunum is sutured across the entire diameter of the stomach buttressing it over the closed portion of the stomach so that the jejunum does not hang by the stoma. To prevent sagging or torsion, the region of the greater curvature is reinforced with omentum and the afferent loop is fixed to the posterior peritoneum and the upper portion of the gastrohepatic ligament with a few interrupted sutures.

The postoperative course is usually uneventful. However, there are cases in which unexpected difficulties will arise. It is the purpose of this presentation to review the more common postoperative complications following this procedure.

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When these occur we should be able to recognize them and handle them properly.

The best treatment, of course, is prevention. To better understand why these complications occur and what steps to take to prevent them is important. Recently I heard this formula for success, "Success comes from good judgement; good judgement from experience; experience comes from bad judgement." I am sure we have all made errors in judgement and striving for perfection should be one of our major objectives.

Duodenal Leakage

All of us have been faced with the problem of the difficult duodenal stump. In many cases our surgical judgement and ingenuity will be taxed. However, this is one of the most important steps in the operation. A blowout of the duodenal stump carries a mortality ranging from 50 to 80 per cent according to Sanford who reviewed the literature in 1956. According to the same author, the incidence of duodenal stump dehiscence is 1.5 to 3.5 per cent and may be caused by several factors which are:

1. Local
   a. Edema and inflammation from the ulcer
   b. Impairment of blood supply
   c. Devitalization of tissue from clamping
2. Systemic
   a. Hypoproteinemia
   b. Anemia
   } Both interfere with healing
3. Obstruction of the proximal loop or stoma causing increase in intraduodenal pressure
4. A combination of factors

We feel that when operating upon duodenal ulcers, particularly bleeding ulcers, they should be excised if possible. There are occasions when, because of the location of the ulcer, it would be wiser to use more discretion than valor and to leave the ulcer *in situ* if the gastric mucosa is excised. If the ulcer is actively bleeding, excision of the ulcer or ligation of the vessel should be carried out. The common duct and the pancreas

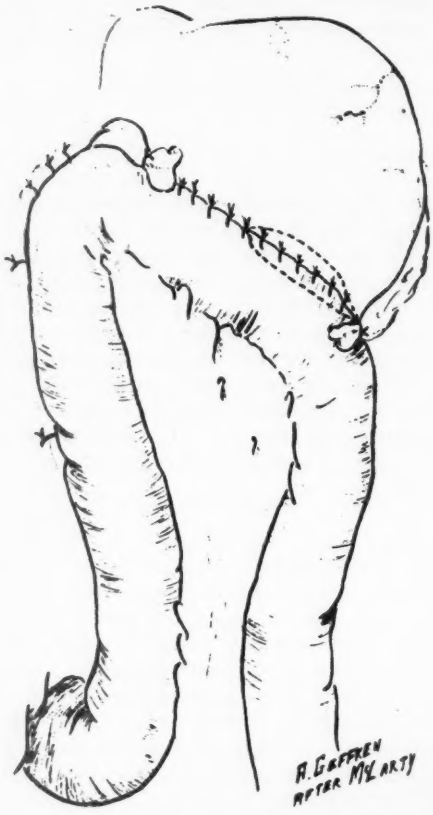

FIG. 1. Subtotal gastrectomy; antecolic isoperistaltic Hofmeister-Polya technique.

must not be injured. The duodenum should be dissected down far enough to obtain adequate length for closure. If there is any question about the common duct it should be demonstrated by a choledochostomy. If the duodenum can be closed but the integrity of the suture line is questioned, the area should be drained with Penrose and sump drains to convert any possible subsequent leakage into a duodenal fistula. We do not drain the abdomen routinely when a secure closure is obtained. However, when there is doubt about the closure, a drain does no harm and should there be leakage a prompt diagnosis can be made, and another operation thereby avoided. In the event it is impossible to close the duodenum, a catheter duodenostomy as advocated by Welch[23] should be done. Larson and Foreman[7] considered a delay in diagnosis a significant factor in the high case fatality rate—75 to 85 per cent reported in the literature.

If duodenal leakage occurs it can lead to peritonitis, subphrenic abscess, or fistula formation. It usually manifests itself in 5 to 7 days postoperatively by right upper abdominal pain, tenderness, sudden rise in temperature and pulse, and sometimes by bile colored drainage through the incision. These signs may be masked by analgesics or thought to be pulmonary in nature unless one is on the alert. An x-ray usually will show pneumoperitoneum (fig. 2).

If drainage has not been provided, early surgery is mandatory or generalized peritonitis will ensue. The abdomen should be reentered and rubber tissue drains placed down to the duodenal stump. No effort should be made to close the duodenum.

The fistula will usually close spontaneously. However, because of excoriation of the skin with its associated discomfort and the loss of electrolytes, the time can be shortened after a sinus tract has been formed by inserting a Bardex catheter into the tract and inflating the balloon. This serves to force the duodenal contents into their proper channel. Careful attention should be paid to fluid and electrolyte replacement in a patient with a fistula.

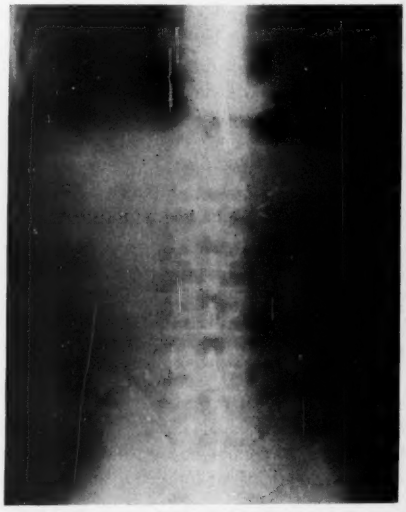

FIG. 2. Roentgenogram of abdomen showing pneumoperitoneum following blowout of duodenum.

Stomal Complications

One of the most distressing complications to both surgeon and patient alike is a gastroenterostomy stoma that will not function. This may occur with either the afferent or the efferent loop.

If the afferent loop is obstructed, biliary and pancreatic secretions are blocked causing dilation of the afferent loop. Usually the duodenal sutures will blow out, but if the duodenal suture line holds, the loop may become tremendously dilated. This should be suspected whenever the gastric aspiration or vomitus does not contain bile. One of the first things we begin to look for 12 to 24 hours after a gastrectomy is the presence of bile in the drainage bottle and we are always relieved to see green biliary drainage. To prevent tension and kinking at the stoma, the proximal loop should not be too short. On the other hand, it should not be too long because it may become "waterlogged" with secretions and have difficulty emptying itself through the stoma.

When the efferent loop is obstructed, the gastric remnant does not empty and unless the contents are aspirated the patient will intermittently vomit large amounts of bile and food. It should be suspected when the patient refuses to eat because of epigastric fullness, nausea, and vomiting. Gastric aspiration will relieve the symptoms and x-ray will show the barium meal to be completely retained in the gastric segment (fig. 3).

Obstruction to the loops may be caused by:

1. Functional conditions
   a. Edema
   b. Hypoproteinemia
   c. Spasm

but usually they are caused by:

2. Organic conditions
   a. Kinked loop
      1. Either too short or too long afferent loop
   b. Adhesions
   c. Constricting ring caused by inverting excessive amount of tissue at the anastomosis
   d. Localized peritonitis with abscess formation; hematoma
   e. Stoma too small or improperly placed
   f. If the retrocolic method is used, the anastomosis may be pulled upward through the transverse mesocolon

The obstruction may be mild, moderate, or severe. The mild type will be relieved by proper attention to fluid, electrolyte, and protein needs.

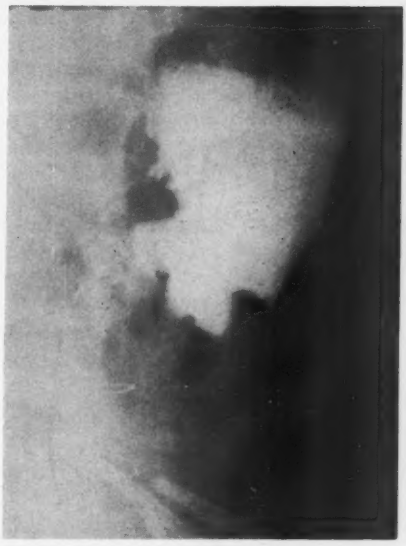

FIG. 3. Roentgenogram of abdomen; gastric retention caused by obstruction of efferent loop.

The proximal loop syndrome characterized by the long dilated afferent loop, as described by Wells and MacPhee,[24] is typical of the moderate type. It may occur in the immediate postoperative period or may occur many weeks later. It is characterized by fullness and discomfort to the right of the midline, nausea, and anorexia associated with a bitter taste in the mouth. Periodically, the patient vomits large quantities of bile. The severe type of obstruction is a surgical emergency and if untreated will result in a fatal closed loop type of obstruction with gangrene. It is usually caused by a long proximal loop passing between the shortened mesentery of the distal loop and the transverse colon.

In mild or moderate obstructions, conservative treatment should be carried out. Just how long to persist must be individualized. It is difficult to remain undisturbed when the patient's veins for parenteral feeding probably have become obliterated and his patience with the clyses and the nasal tube is near the breaking point. Fortunately, most obstructions subside within a few days to a week and one should not reenter the abdomen too soon. A jejunostomy might be done but in recent years we have been using the Puestow-Olander double lumen tube. The gastric portion of the tube keeps the stomach empty when connected to continuous suction while the

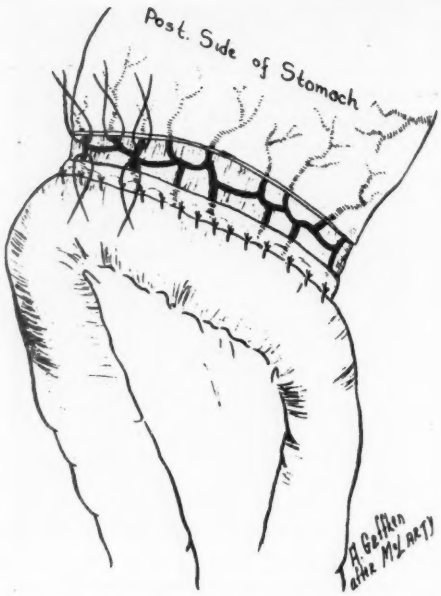

FIG. 4. Schematic drawing demonstrating large gastric vessels which should be meticulously ligated.

feedings, if the stoma is slow to work, can be given through the distal end which projects 30 cm. into the upper jejunum. It also serves as a "splint" to the efferent loop and helps to prevent angulation of this loop.

If the obstruction persists, the abdomen must be reentered. On some occasions, no cause for the obstruction will be found but the anastomosis must either be revised, or a jejunoplasty or an enteroenterostomy must be carried out.

Hemorrhage

Wilkins, Rogers, and Straehley[25] recently reviewed the literature and in a series of 14,324 subtotal gastrectomies they found the incidence of serious postoperative hemorrhage to be about 1 per cent and that 1 patient in 10 having such a complication may be expected to die from it.

The sources of the bleeding are usually: (1) The anastomotic suture line or duodenal suture line; (2) a remaining or overlooked ulcer; (3) postgastrectomy gastritis; (4) new gastrojejunal ulcer—occurs 3 to 4 weeks postoperatively; (5) hemorrhagic disease; (6) jejunogastric intussusception.

It is felt that the most common site of the bleeding—from the anastomotic suture line—is caused by a technical error at the time of surgery. British observers have shown that there are arteriovenous communications in the vascular bed of the stomach wall and therefore these vessels have a high pressure and bleed profusely when the stomach is sectioned. Consequently, the large and numerous gastric vessels (fig. 4) should be meticulously ligated, preferably by an open anastomosis. Hemostatic rubber shod clamps probably should be avoided as they may temporarily occlude a vessel which might bleed later. The jejunum should be opened at its antimesenteric border—the least vascular area. The inner layer of sutures should be locked for hemostasis.

Treatment of the postoperative hemorrhage should be approached conservatively at first with blood replacement and gentle lavage and suction of the gastric stump. The use of absorbable gelatin sponge powder (Gelfoam) is of doubtful value but vitamin K, calcium, and fibrinogen must be considered if the clotting mechanism is deficient. In the rare case which is not controlled by conservative means, the patient must be explored.

If the bleeding is from the anastomotic suture line, a running stitch usually suffices; if from the duodenum, this must be reopened; if from a bleeding ulcer, the ulcer must be excised, the pancreaticoduodenal artery ligated, or absorbable Gelfoam or Oxycel packing used. Bleeding as late as 7 to 10 days postoperatively from sloughing at the suture line will usually stop spontaneously and only rarely calls for surgical intervention.

Pancreatic Complications

The pancreas must constantly be kept in mind both during surgery for subtotal gastrectomy and in the postoperative period. Injuries may cause a severe pancreatic reaction or pouring out of secretions causing various complications. Prevention is the best treatment and injuries to the pancreas should be avoided by gentleness and meticulous surgical technique. Ulcer bases should not be excised. Should injuries to the pancreas occur, they must be recognized and repaired immediately.

Acute pancreatitis postoperatively is a complication which has been receiving more attention in recent years. It should always be thought of in a postoperative gastric patient who develops upper abdominal pain, distension, pain in the

back, elevated pulse and temperature. A serum amylase should be obtained early to make the diagnosis because medical management is the treatment of choice. A dangerous and unnecessary exploratory laparotomy at this time increases the mortality rate.

Dumping Syndrome

After a subtotal gastrectomy many individuals complain of epigastric fullness, discomfort, weakness, nausea, sweating, tachycardia, dizziness, and even collapse when they resume feeding. The cause of these symptoms is poorly understood but they are generally classified as the "dumping syndrome." The reported incidence is 3 to 80 per cent depending upon the interpretation of the symptoms. No technique of gastrectomy will insure freedom from the development of such symptoms but fortunately they improve with time and in most cases completely disappear. With the technique described previously, we have had very little difficulty with the dumping syndrome and in most cases the eating capacity soon returns to the preoperative level. The symptoms may be divided into two main groups depending upon whether they appear early or several hours after a meal. The early symptoms come on a few minutes after completion of the meal and are thought to be mechanical in origin. The later symptoms occur 2 to 3 hours after eating and are thought to be caused by hypoglycemia.

Various theories have been developed to explain this syndrome, namely:

(1) Overdistension of the jejunum from too rapid emptying of the gastric remnant. Similar symptoms can be caused by too rapid jejunostomy feedings. (2) Dragging down of the gastric remnant by an overfilled jejunum. (3) Stasis in the proximal loop. (4) Overdistension of the small intestine as a result of the increased volume of fluid which enters the gut lumen in an attempt to render the osmotic properties of the ingested food isotonic. (5) Too rapid absorption of carbohydrates which gives rise to hyperglycemia, later followed by hypoglycemia, and giving rise to the same symptoms as hyperinsulinism. This usually occurs about 2 hours after eating and is known as the delayed dumping syndrome.

Undoubtedly, the dumping syndrome is dependent upon a number of factors that vary in different cases because of the varied methods of treatment that have given relief. Accepted treatment includes small, frequent meals; omission of sweets, carbohydrates, and fluids at mealtime for the early syndrome. Some advocate lying down after meals and eating fats to slow gastric emptying. For the later syndrome, it is wise for the patients to carry candy or sweets around with them to eat when the premonitory symptoms appear. Surgery is indicated only in cases proven to have the proximal loop syndrome but some authorities feel that too rapid emptying of the gastric remnant is an indication for making the stoma smaller.

Recurrence of Ulcer

One of the most discouraging complications after subtotal gastrectomy for duodenal ulcer is a recurrent ulcer which occurs in about 5 per cent of cases. These usually occur on the jejunal side of the anastomosis and are manifested by ulcer symptoms, hemorrhage, obstruction, and perforation as well as gastrojejunocolic fistula.

It is thought that an inadequate resection in which the acid secreting ability of the stomach is insufficiently reduced is the chief cause of recurrent ulcer. They rarely occur following resection for gastric ulcer. Treatment consists of further resection of the stomach and vagotomy if an inadequate procedure has been done, or a vagotomy if a higher resection is not indicated.

Inanition

Another undesirable end result in the postgastrectomy patient is inanition. Some individuals, particularly those underweight preoperatively, never get back to their preoperative weight level. The best results from the standpoint of nutrition are obtained in patients who have been somewhat overweight at the time of surgery and seem to regain their weight postoperatively.

Factors that may be involved are: (1) Amount of stomach removed; (2) rapid transit of food through the gastrointestinal tract; (3) impairment of fat digestion and absorption; (4) poor mixing of bile and pancreatic juices with food; (5) dietary restrictions because of symptoms of dumping, etc.; (6) emotional factors.

Anemia is frequently seen in gastrectomy patients, particularly in females. It is usually microcytic in type and responds readily to iron therapy. Some physicians recommend intermittent administration of iron to all postgastrectomy

patients and a hemoglobin determination at least once a year for the rest of their lives. Primary anemia has been reported but is rare.

Other Complications

1. Wound dehiscence—occasionally occurs and must be treated with through and through closure.

2. Thrombo-embolism—still the enigma of the surgeon though less common since early ambulation; treated by anticoagulation therapy, vein ligation, or both.

3. Wound infections—greatly reduced since the era of antibiotics.

4. Serum hepatitis—usually from blood transfusions.

5. Pulmonary complications—with modern anesthesia and antibiotics is much less common than previously.

6. Allergies to drugs—exfoliative dermatitis from penicillin or other antibiotics may occur.

7. Intraperitoneal infection—peritonitis is rare unless there is a leak from the anastomotic suture line. This shows up between the fifth and twelfth day but reintervention may be necessary. Favorite sites for secondary abscesses are in the subdiaphragmatic and subhepatic areas.

8. Intraperitoneal hemorrhage—rare but should be considered in any patient with tachycardia and hypotension after gastrectomy; all major blood vessels as right and left gastric, etc., should be doubly ligated.

9. Obstruction: (*A*) Intestinal obstruction from adhesions. (*B*) Internal hernia—may occur a few days postoperatively; symptoms of high obstruction; emergency exploration is indicated. (*C*) Jejunogastric intussusception—rare but occurs occasionally regardless of type of anastomosis; etiology is undetermined; symptoms of high obstruction; immediate operation with reduction indicated.

10. Laceration of the spleen—usually from retractors.

11. Ischemia of the colon—occurs from injury to the middle colic artery; segmental resection of transverse colon indicated.

12. Ischemic necrosis of gastric remnant—rare.

13. Gangrene of the greater omentum—rare.

14. Obstructive jaundice and bile peritonitis—may occur as a result of surgical trauma or previous disease. Laparotomy is necessary with drainage of the gall bladder, common duct, or peritoneal cavity.

15. Incisional hernia—may occur in elderly or poorly nourished individuals. Also frequently follows in patients who have poor wound healing because of exsanguination, prolonged stomal obstruction, anorexia, and inadequate diet.

SUMMARY

1. Subtotal gastrectomy remains the surgical treatment preferred by most surgeons for peptic ulceration.

2. A high gastric resection and an antecolic Hofmeister-Polya isoperistaltic anastomosis is advocated.

3. Postoperative complications must be carefully watched for and skillfully treated.

4. Duodenal stump leakage occurs in 1.5 to 3.5 per cent of cases and carries a high mortality.

5. Duodenal ulcers should be excised if possible; care must be taken to prevent injury to the pancreas and common bile duct.

6. When the duodenal stump cannot be securely closed, drainage of the peritoneal cavity must be provided.

7. The afferent or efferent loop may become obstructed; stomal obstruction may be mild, moderate, or severe.

8. Postoperative hemorrhage occurs in 1 per cent of cases; treatment of postoperative hemorrhage should at first be conservative with blood replacement; exploration may be indicated.

9. The dumping syndrome occurs in some cases but fortunately symptoms tend to disappear in time. With the operative technique described, dumping symptoms have been minimal.

10. Inanition, pancreatic complications, recurrence of ulcer, and other less common complications are discussed.

735 Piedmont Ave., N.E.
Atlanta, Ga.
(*Dr. Evans*)

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Book Review

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

Anomalies of Intestinal Rotation and Fixation. By R. L. ESTRADA, M.D., F.A.C.S., R.R.C.S. 161 pp., Charles C Thomas, Springfield, Ill., 1958.

The introduction to this interesting and easily read book deals with a discussion of the normal embryology of the alimentary canal in which the author also proposes a standard classification of nomenclature which he feels is essential. He makes a plea for adoption of the term "mesenteric oparietal hernia" in place of the less descriptive "paraduodenal" or "retromesocolic" hernia. Nonrotation, mixed rotation, and reversed rotation are classified and described as separate entities. The substitution of these definitive terms in place of the more commonly used "malrotation" is urged.

In subsequent chapters, the embryologic development and the clinical considerations of each of the conditions are described in a concise and clear manner. The stages of development are divided into (1) herniation with substages deviation and realignment, and (2) reduction and fixation.

The anomalies are grouped according to the abnormal stage. The descriptions are further enhanced by illustrative drawings. Twenty-nine representative case histories from the author's experience, as well as from original reports of the extremely rare conditions, are presented, with accompanying radiographs and pathologic findings. Dr. Estrada describes his techniques for surgical correction of left mesentericoparietal hernias in detail and comments on operative procedures recommended for other anomalies. An outlined summary of the context is included and an extensive bibliography of over two hundred references is available.

Heretofore, most of the literature discussing these problems has consisted of case reports and an occasional review of a particular subject. The difficulty of organizing material on all of the known anomalies in a single monograph is obvious. However, this task has been met by the author with a good degree of success. It should prove worthwhile reading to practicing surgeons and a valuable aid to surgical residents.

JAMES M. CHASE, JR., M.D.

Editorial

SURGICAL TRUSTEESHIP—A MUST!

The insurance buying public and the health insurance companies still have a choice of roads that lead away from governmental control of medicine, but these roads need some medical and surgical safety markers. We should analyze and compromise, where possible, the objections offered by the public, the medical profession, and the insurance companies and make certain that the system of voluntary health insurance continues to grow.

Many working groups would like a "full service" plan to include all medical care with a fixed fee for each service, but, except in localized instances, the cost would be prohibitive on an individual basis. At the other extreme, there is the rapidly growing "major medical comprehensive" coverage which embodies a deductible feature either in dollars and/or percentage shared by the insured and the underwriter. As experience with group coverage of "major medical" increases, individual coverage may soon be obtainable at the same premium rate available now to large groups.

Although the increasing number of insurance forms to be filled in and signed are sometimes irksome, all of us are glad that more and more people have some type of health insurance. Since most families are mainly concerned with hospital, surgical and maternity benefits, we, as surgeons, occupy an important spot in the picture. What are some of our responsibilities? First, we should support, *full service* plans designed for those people who are neither indigent nor affluent. Most states have such a fixed fee for service plans which covers families with incomes up to an average of $4200 annually. Such plans should be fairly regulated, so that patients with multiple health insurance policies and those whose incomes exceed the maximum limit may expect to pay the surgeon's customary fee instead of the more limited service fee. Many states have optional service benefits to cover in-hospital medical care and the radiologic treatment of malignant lesions. Thus individuals and groups eligible for these lower fees for service can obtain adequate coverage at reasonable cost where some could ill afford the deductible feature of the more complete coverage plans.

"Major medical" expense insurance is a very rapidly growing eight-year-old, and it should appeal to all physicians and surgeons, because there is no fixed fee for any service. It represents a new concept and a successful effort on the part of the insurance companies to meet the need for protection against large hospital and medical bills of all kinds, including nursing care. The number of people covered by major medical policies multiplied fourfold in the last two years, and both group and individual policies are available with more companies. The principles of coinsurance and a fixed deductible are a basic part of major medical coverage and permit broad coverage of medical expenses and a high maximum to care for "catastrophic" accidents or illness.

How can we, as surgeons, help these plans grow? We can help educate our patients to the advantages of good policies but, most important, we can help limit the rising costs of medical care by carefully evaluating our services and making a fair and just charge. We should remember that insurance companies do not create money but rather are trustees for their policy holders and that doctors have a joint trusteeship with the companies. If fees exceed the "usual and customary charge" for that locality, then the premium must be increased and protection would be available to fewer people.

We must protect the vast majority of doctors and patients against the rare doctor who charges an exorbitant fee and the rare patient who urges his doctor to make the charge to the insurance company high enough to include the patient's proper percentage due the doctor.

Much of the controversy over Medicare would be resolved if it could be shown that surgeons can and will limit fees to the "usual and customary" charge for the service rendered. Discussions at local, state and national surgical societies should be helpful in preserving systems in which we believe.

James A. Kirtley, Jr., J.D.
Chairman, State Prepaid Insurance Committee
2318 West End Avenue
Nashville, Tennessee